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Archives of Medicine

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DEVOTED TO ORIGINAL COMMUNICATIONS ON MEDICINE,
SURGERY, AND THEIR SPECIAL BRANCHES

EDITED BY
E. C. SEGUIN, M.D.

> S'il est possible de perfectionner l'espèce humaine, c'est dans la médecine qu'il faut en chercher les moyens.
>
> —DESCARTES

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ARCHIVES OF MEDICINE.

Original Articles.

THE MYOGRAPHY OF NERVE DEGENERATION IN ANIMALS AND MAN.*

By R. W. AMIDON, A.M., M.D.,
NEW YORK.

IN as few words as possible, the object of this paper is to study, by means of careful electrical examinations and myographic tracings, the effects of peripheral nerve injury in man; alongside of which to place similar observations on frogs whose nerves have been cut, and, by knowing the histological changes occurring in the frog, to infer that not dissimilar ones exist in man when the outward manifestations are the same.

In these days, so prolific in medical literature, only the shorter, more practical articles receive careful perusal, and it is in the endeavor to make this article within the reach of all, that speculation and theorizing, details and useless histories of typical cases, will be omitted.

As a sphygmogram is a pulse-tracing, so a myogram is a muscle-tracing. By means of a mechanism, to be described later, a muscle, by its contraction, moves a lever which makes a mark on the smoked paper of a revolving cylinder. It writes indelibly on paper the following facts: quickness of muscular contraction and relaxation (abruptness of the up- and down-stroke), amplitude of the muscular movement

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* Read at the meeting of the American Neurological Association, June 21, 1882.

(height and breadth of curve), and, by means of some accessory apparatus (the chronograph and the tuning-fork), the quickness of contraction after the application of stimuli (the latent period), and the effect of a continuance or a withdrawal of the same.

Any one at all conversant with electro-therapeutics will recall the retarded, slow, vermicular contractions which galvanism excites in muscles which have been cut off for some time from the spinal cord. The peculiarity of this contraction the writer thought could be beautifully demonstrated by the myograph, and it was with the idea of imitating this condition in the animal that the following experiments were performed.

In the first place normal nerve and muscle reactions were taken in healthy frogs as follows. By means of a double interruptor the circuit carrying electricity to the frog is made and broken at the same time as the circuit of the chronograph, hence the moment the electricity reaches the muscle it also reaches the chronograph and breaks the line.

Having broken up the brain and spinal cord of the frog, to stop voluntary and reflex movements, the sciatic is exposed, the tendo Achillis is cut and fastened to the myograph needle, and the frog is securely pinned to the cork stage (see fig. 1), with one electrode under the nerve and the other in some indifferent point, as the small of the back. We take the tracings produced by the cathode (neg.) closure contraction, cacc, and the anode (pos.) closure or opening contraction, ancc or anoc, excited by a small Grenet cell, and afterward the reaction to the secondary current of a Dubois induction apparatus. The same process is repeated with the muscle.

The sciatics of several frogs were cut and the animals were kept in a *very* warm room, so that nerve degeneration would not be retarded for perhaps weeks, as it sometimes is in frogs, especially in the winter.

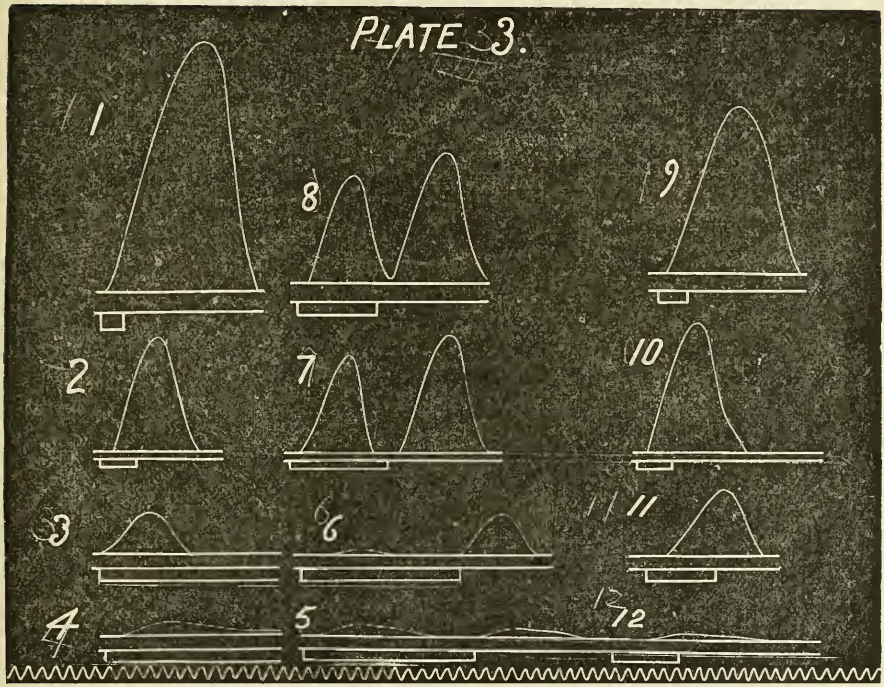

PLATE iii.—Myograms from excitation of the nerve in the frog.

FIG. 1.—Normal cacc ; fig. 8, normal ancc and oc ; fig. 9, normal fc, with secondary coil at 7 centimetres.
FIG. 2.—Cacc ; fig. 7 ancc and anoc ; fig. 10 fc, on second day of section, " " " " "
FIG. 3.— " fig. 6 " " " fig. 11 fc, " third " " " " " " " "
FIG. 4.— " fig. 5 " " " fig. 12 fc, " fourth " " " " " " " "

The line on which the curves stand is called the base line, and is made by the myograph needle when at est. The straight line below and parallel to this is made by the chronograph needle at rest, and the deviations rom it denote the duration of the passage of the electrical current. The wavy line underneath is made by a uning-fork vibrating one hundred times to the second.

Each day a frog was killed, pithed, and put through the same routine treatment as the healthy ones had been. After the electrical examination, a microscopic study of the injured nerves and muscles was also made. The formula for normal nerves is,[1] cacc > anoc > ancc > caoc (the last being seldom or never seen in healthy nerves). This formula is graphically represented in pl. iii, figs. 1, 8, and 9, where the amplitude of cacc is > anoc, which is > ancc, and where good muscular contraction is caused by the induced current of Dubois' secondary coil at a distance of seven centimetres from the primary coil (a current just perceptible on the tongue).

The changes in the nerve reactions following section were as follows: Forty-eight hours after section decided changes had taken place; cacc had diminished two thirds, anoc and cc were about the same, and faradic reaction was diminished about one third. On the the third day cacc$=\frac{1}{6}$, anoc$=\frac{1}{4}$, ancc just perceptible, and fc $\frac{1}{3}$ its normal amplitude. On the fourth day cacc was diminished to $\frac{1}{18}$th, slow and tetanized; anoc was diminished to $\frac{1}{12}$th, ancc the same size, and both very slow and tetanized; fc about $\frac{1}{16}$th, retarded, slow, and tetanized.

On the fifth day of section no nerve reaction to galvanism or faradism could be detected. The Wallerian degeneration began to be very distinctly discoverable, on microscopic examination of the nerves, on the third day, increased each day, and on the fifth, and afterward, was complete, the nerves being one mass of broken myeline matter, nuclei, granular detritus, and naked axis-cylinders. This series of tracings demonstrates very beautifully the gradual death of a degenerating nerve, before only shown by the progressive diminution of electrical reactions as visible to the unassisted eye. The nerve died early in these frogs because of the warm temperature in which they were kept. In man the nerve ceases to respond, if cut or badly bruised, on the 7th to the 14th day.

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[1] Hereafter the customary abbreviations will be used: ca. cathode (neg. pole), an, anode (pos. pole), c = closure, o = opening, c = contraction, > = greater than, < = less than, = equal, f = faradism, g = galvanism, etc.

The muscles supplied by a degenerating nerve pursue a different course. The normal formula produced by direct excitation of the muscle is cacc>ancc>anoc>cacc (the two last being rarely seen). This formula is graphically represented in pl. iv, figs. 1, 26, 27, where cacc is one third greater than ancc, and where fc is produced by the secondary coil of Dubois at 4 cm. distance. Now, a word as to the degeneration of nerves in man. In the first three to four days all the electrical excitants produce simply less than normal reaction. Later (3d-7th day) fc gradually diminishes, and disappears in the second week. Ancc=cacc, and in second or third week becomes >cacc. Not only this, caoc rarely, or, we may say, never seen in healthy muscle, now appears. Thus the formula is often reversed, ancc>cacc>caoc>anoc (the last seldom seen). The appearance of these *qualitative* changes indicates the arrival of the Wallerian degeneration at the termini of the motor nerves in the muscle. From this time on the connective tissue increases in the muscle, cirrhosis ensues, ancc becomes very much>cacc, caoc and cacc gradually disappear, and, last of all, when all muscular tissue has disappeared, ancc also is wanting. Again, muscles contract more and more slowly when degenerating. Their contraction is retarded (increased latent period) and prolonged, and, in certain cases, a tetanized contraction can be produced by a continued galvanic current, which, in healthy muscle, can only be produced by a *very* strong current in a fatigued or excited muscle.

The frog's muscles after excision of the sciatics were examined as follows: Each day a new frog was killed, pithed, and tested.

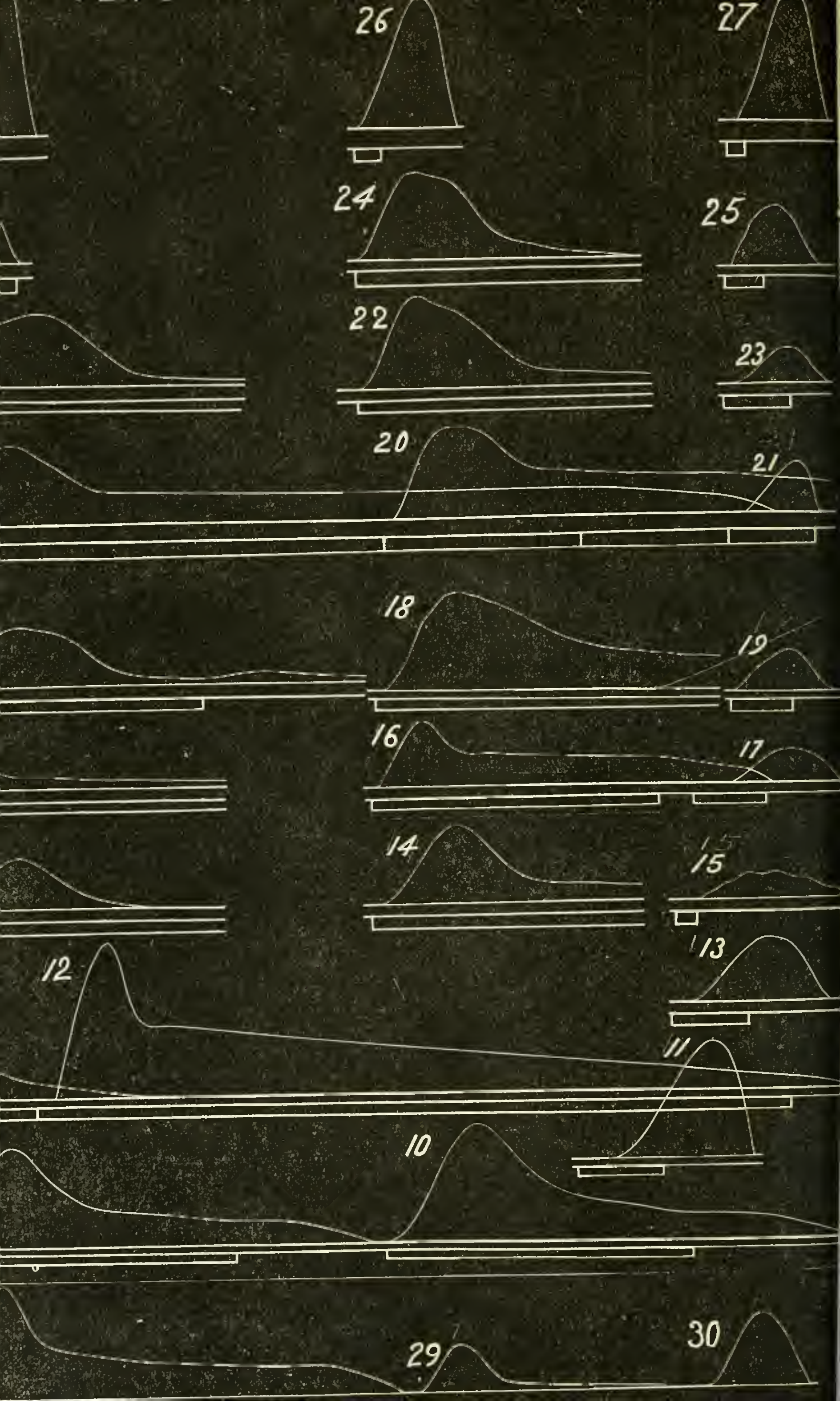
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PLATE iv.—Myograms produced by direct stimulation of frog's muscles before and after section of the sciatic nerves.

FIG. 1.—Normal cacc 1 cell; fig. 26, normal ancc 1 cell; fig. 27, fc, with secondary coil 4. c. distant.

FIG. 2.—2d day of section cacc, 1 cell; fig. 24 normal ancc, 1 cell; fig. 25 fc, with secondary coil 4. c. distant.

FIG. 3.—3d day of section cacc, 1 cell; fig. 22 normal ancc, 1 cell; fig. 23 fc, with secondary coil 4. c. distant.

FIG. 4.—4th day of section cacc, 1 cell; fig. 20 normal ancc, 1 cell; fig. 21 fc, with secondary coil 4. c. distant.

FIG. 5.—5th day of section cacc, 1 cell; fig. 18 normal ancc, 1 cell; fig. 19 fc, with secondary coil 4. c. distant.

FIG. 6.—7th day of section cacc, 1 cell; fig. 16 normal ancc, 1 cell; fig. 17 fc, with secondary coil 4. c. distant.

FIG. 7.—16th day of section cacc, 5 cells; fig. 14 normal ancc, 1 cell; fig. 15 fc, with secondary coil in contact.

FIG. 8.—20th day of section cacc, 1 cell; fig. 12 normal ancc, 1 cell; fig. 13 fc, with secondary coil in contact.

FIG. 9.—37th day of section cacc, 5 cells; fig. 10 normal ancc, 1 cell; fig. 11 fc, with secondary coil in contact.

FIG. 28.—44th day of section cacc, 5 cells; fig. 29 normal ancc, 1 cell; fig. 30 fc, with secondary coil in contact.

Line at bottom marking $\frac{1}{100}$ths of a second.

First, a curve was produced by cacc, then by ancc, then by an induced current from Dubois' secondary coil 4 centimetres distant. Forty-eight hours after section there was already diminished amplitude of muscular movements, shown by lower curves, and already ancc > cacc, while there was a commencement of tetanization in the anode curve, *i. e.*, the down-stroke was slanting and prolonged (pl. iv, fig. 24). On the third day ancc was > cacc, while fc was very slight indeed; cacc also showed tetanization, the curve being rounded and prolonged long after the electric stimulus was withdrawn. The latent period[1] was already lengthened (the normal duration of the latent period being about $\frac{1}{100}$ second). In certain pathological states[2] this period is much prolonged, and the time elapsing between the entrance of the electricity into the muscle and its contraction, is visible to the eye, but much better by aid of a myogram. This retardation is particularly marked in muscles showing degeneration reaction.

On the fourth day tetanization was still more complete, the muscle not relaxing at all during the continuance of the current, and not even till several hundredths of a second after the opening of the circuit.

On the sixth day cacc is smaller, and there appears also a small caoc; ancc is larger and very tetanic.

On the seventh day cacc is small ($\frac{1}{6}$ the amplitude of the normal) and tetanic; ancc is $2\frac{1}{2}$ times > cacc.

On the sixteenth day, pl. iv, figs. 7, 14, 15, there is hardly a trace of cacc with one cell, while five cells produced a slow contraction retarded $\frac{5}{100}$ second, while the single cell still produced a large and tetanized ancc, *i. e.*, ancc was ten times > cacc. Fc was only obtained by bringing the secondary coil of Dubois in contact with the primary coil, producing a current which threw the frog into general tetanus, the current thus produced being almost painful to the operator's hand.

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[1] The latent period is the time elapsing from the excitation of the muscle to its contraction.

[2] Mendelssohn: Sur le temp perdu des muscles. Physiol. experiment. (Marey), Paris, 1880.

On the twentieth day there was a small cacc (pl. iv, fig. 8) to one cell, while the anode of the same cell excited an immense tetanized curve. A fair fc, which is retarded, was produced by the secondary coil at contact.

On the thirty-seventh day the myographic curves presented peculiarities which, as examples to follow will show, must be considered as pathognomonic of the degeneration of nerves. From the first it has been shown that the down-stroke was getting more and more slanting and slow to reach the base line ; *i. e.*, the muscle was slow to relax, and partly tetanized. Now, on the thirty-seventh day, and later, tetanization becomes complete, *i. e.*, so long as the galvanic current is allowed to pass through the muscle, it will not relax, but as soon as the circuit is broken, a slow relaxation takes place. This is shown in figs. 9, 10, and 28, pl. iv, where we have a sloping up-stroke, a rounded top, a sloping down-stroke to a certain point where it remains stationary until, when the circuit is broken, it gradually falls to the base line.

This series of myograms shows graphically the progressive changes, quantitative and qualitative, which an irreparable injury to a motor nerve produces in the tributary muscles. First, the retarded, tetanized, and gradually vanishing cacc ; secondly, the retention of ancc, which, however, is also retarded and tetanized ; thirdly, the preponderance of ancc over cacc ; and lastly, the rapid failure of faradic contractility ;—the later seemingly large contractions to faradism being the results of the application of a stimulus ten or more times the strength of the current first used. The reason that fc is retained at all is because the electrode comes in direct contact with the muscle, the same thing occurring in man if the muscle be punctured. The only histological change noticed in the muscles of the frogs experimented on, was a granular, disintegrated condition of the intramuscular nerves.

The myographic studies undertaken in man[1] were chiefly in cases of nerve injury, or diseases which cause nerve-degeneration and muscular atrophy. The same appliances for registering were used in man as in frogs, but in a way the accompanying diagram will illustrate.

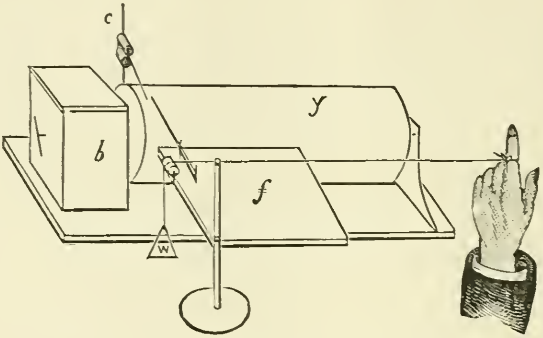

FIG. I.

MODIFIED MYOGRAPH FOR EXPERIMENTATION ON MAN.

b.—Clock-work with revolving cylinder, *y.*
c.—Chronograph apparatus (coils and needle).
f.—Frog-plate of cork, with its pulley and weight (w), and its writing lever, to which the movable part to be experimented on is attached by an inextensible thread.

The manner of procedure, suppose we be experimenting on the abductor indicis, is as follows. Place the hand on a rest of some kind at the same level as the frog-plate. Fix one electrode on an indifferent part of the body (preferably the sternum). Bind another small electrode over the ulnar nerve at the elbow or wrist, connect one directly with the battery, and the other with the battery through the mediation of the double interruptor. Turn on enough cells to produce good cacc, set the cylinder going, and register several curves. Mark them *cacc*, and the number of cells used. Reverse the current, and register ancc the same way. Remove the cords from the galvanic battery, and connect with Dubois' secondary coil, and register fc the same way, noting the relations of the two coils (strength of current).

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[1] *Mention* of human myography can be found in Cyon's "Principes D' Électrothérapie," and in some of Marey's books, but the author has nowhere seen myograms from man, and but few from *any* mammal.

To take muscular reactions, leave the indifferent electrode on the same part, and bind or hold a small electrode on the motor point of the muscle to be tested.

In pl. ii, figs. 1, 16, 17, are seen healthy human myograms, produced by excitation of nerves, and the attention of the reader is particularly called to the similarity between them and those of the frog, the only difference being that in this subject the ancc could not be brought out on account of the great pain caused, and the excitation of antagonistic muscles. In speaking of pathological myograms the reader's time will be economized by giving the *name* of the disease studied (only typical, indubitable cases being employed), and other such data as are essential to a correct understanding of the subject.

In Bell's palsy of the face we have a disease very closely resembling nerve section, a resemblance which, in incurable cases, amounts to an identity as regards pathological changes and external phenomena. It matters not how muscles are severed from their centres, whether by neuroma, neuritis, compression, contusion, or section, an identical result will follow an irreparable injury, viz.: degeneration of the nerve and muscular atrophy. For this reason the accompanying case of Bell's palsy is a good one to place alongside of our frogs whose nerves were cut.

The mode of recording facial muscular movements was by means of connecting the cheek, in the neighborhood of the angle of the mouth, with the myograph, by means of a piece of isinglass plaster, stuck to the skin, from which a

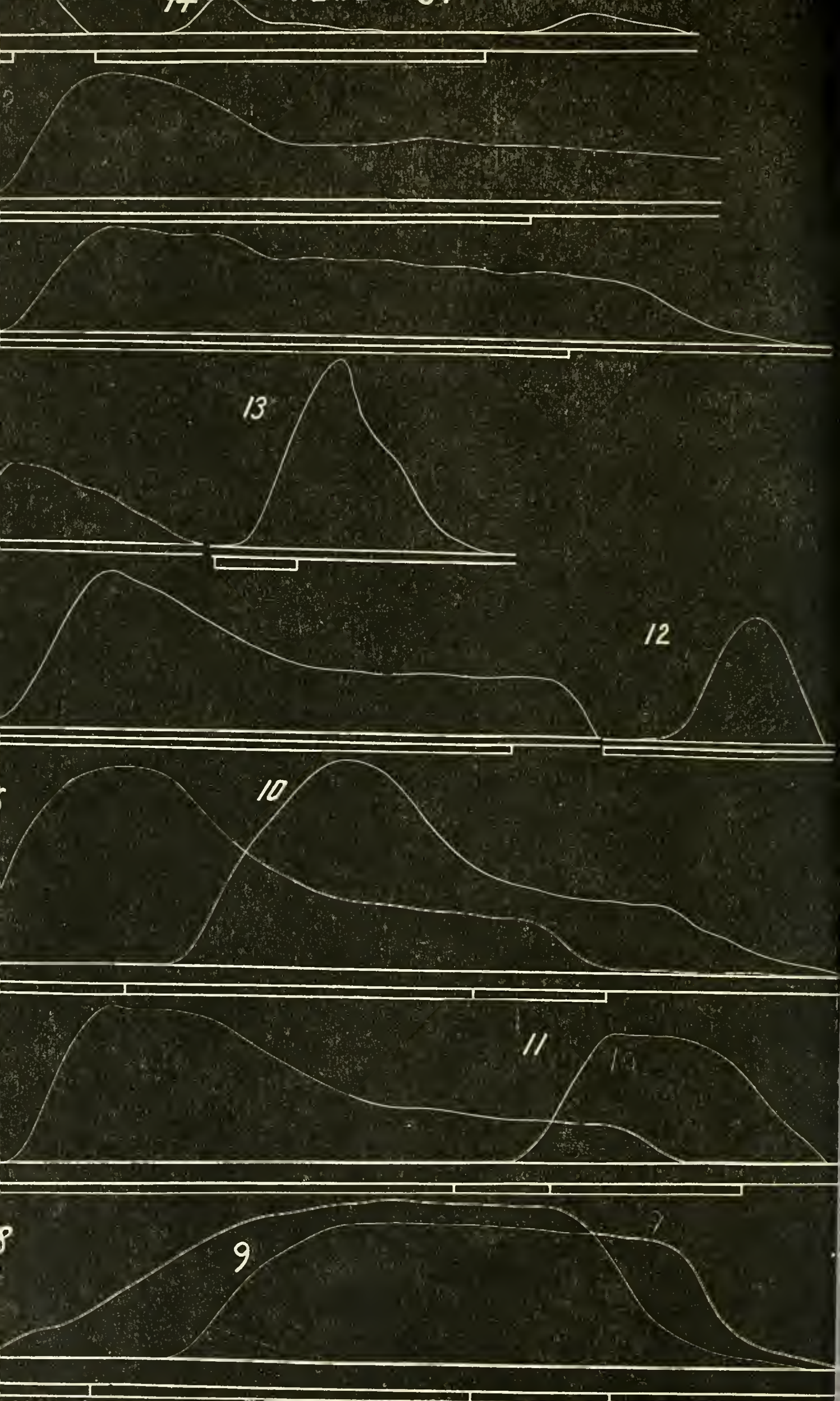
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PLATE vi.—Human myograms, reduced one third. Case of Bell's palsy, etc.

FIG. 1.—Cacc 32 cells, and fig. 14, ancc and anoc 32 cells, nerve current, 14th day.

FIG. 4.—Cacc 25 cells, and fig. 13, ancc 25 cells, muscle current, 14th day. Ancc > cacc.

FIG. 2.—Cacc 40 cells, and fig. 3, ancc 35 cells; nerve current, 31st day.

FIG. 5.—Cacc 20 cells, and fig. 12, ancc 12 cells; muscle current, 31st day. (Ancc 12 cells very much > cacc 20 cells.)

FIG. 6.—Curve produced by extensors of the hand in a case of paralysis, produced by a blow on the musculo-spiral nerve, twenty-six days before. No nerve reaction, cacc 17 cells; fig. 10, ancc 40 cells, cacc > ancc; prognosis good.

FIG. 7.—Another case of paralysis of the musculo-spiral nerve from the kick of a horse. No nerve reactions, 60th day, cacc 40 cells; fig. 11, ancc 40 cells, cacc > ancc.

FIG. 8.—Cacc 47 cells, and fig. 9, ancc 40 cells, in a case of injury to the ulnar nerve by reducing a dislocated humerus with the heel in the axilla. Tracings from direct excitation of the abductor indicis muscle on the 60th day of the injury.

thread was carried to the lever. Then the head was placed so that, the thread being taut, any slight facial movement was recorded on the smoked paper.

The first examination was made in the second week of the paralysis. The formula then stood:

Nerve, cacc > anoc.
Muscle, ancc > cacc.
No fc, nerve or muscle.

All contractions were much retarded and prolonged, the latent period of ancc and oc being about $\frac{7}{100}$ second. (See pl. vi, fig. 14.)

On the thirty-first day a second examination was made, when degeneration reaction was much more marked. The nerve was still excitable to galvanism however. The formula stood:

Nerve, ancc 35 cells about = cacc 40 cells;
Muscle, ancc 12 cells about = cacc 20 cells,

thus showing a very well-marked degeneration reaction. The latent period of cacc, nerve current, was $\frac{15}{100}$ second; that of ancc, nerve current, about the same (pl. vi, figs. 2 and 3), while the latent period of direct muscular excitation was $\frac{10}{100}$ second. Please compare trace 5, pl. vi, cacc in Bell's palsy on the 31st day, with fig. 9, pl. iv, cacc of palsied frog on the 37th day. The similarity, not to say *indentity*, in the two curves is striking. Is it unreasonable to suppose that much the same histological changes were present in both? In injury to the musculo-spiral nerve we have a peripheral palsy practically identical to Bell's palsy and that of nerve section in the frog; therefore compare trace 6, pl. vi, cacc 17 cells and 10, ancc 27 cells, myograms in a case of musculo-spiral paralysis of the twenty-sixth day, with the trace of Bell's palsy on the 31st day (pl. vi, fig. 5), and the frog's myogram (pl. iv, fig. 9) on the thirty-seventh day. A second case of musculo-spiral paralysis at the 60th day (pl. vi, fig. 7) may be added to the list, and a comparison of it with the four previously designated traces needs no word of comment.

There is a striking similarity in these myograms. All are *delayed*, have a *sloping* up-stroke, a *rounded* top, a *sloping* down-stroke to a certain point where the muscle remains *tonically contracted* till a *fall* occurs, about as soon after the *breaking* of the current as it commenced after the *making*.

There are, however, nervo-muscular disturbances in man which it is difficult, or impossible to reproduce in animals, and it will be in the briefest possible way that the myograms of various pathological muscles, chiefly the atrophic, are brought to the reader's notice. The reaction in these cases is almost invariably that of degeneration, *i. e.*, ancc > cacc, no fc and no nerve reaction, galvanic or faradic. In them also the reaction appears slow, worm-like, or wavy to the naked eye, which characteristics are magnified by the myograph.

Take, for instance, an old case of section of the median nerve, pl. i, figs. 6 and 11; a simple atrophy of the first interosseous muscles, pl. i, figs. 7 and 10; a case of functional atrophy of the first interosseous muscles from occupation, pl. v, figs. 6 and 7; an atrophy of the hand, non-progressive and with typical degeneration reaction, pl. ii, figs. 7 and 8. Take also the hand of chronic myelitis of the anterior horns in the adult, with an atrophy located in the first interosseous space, in this respect resembling the atrophy of progressive muscular atrophy, but in others entirely differing from it, chiefly by the fact that it has typical degeneration reaction, see pl. i, figs. 8 and 9, where ancc (25 cells) is 4–5 times > cacc (30 cells). True degeneration reaction is also shown in traces 5 and 8, pl. v, where we have myograms of the abductor indicis, in a case of

PLATE I

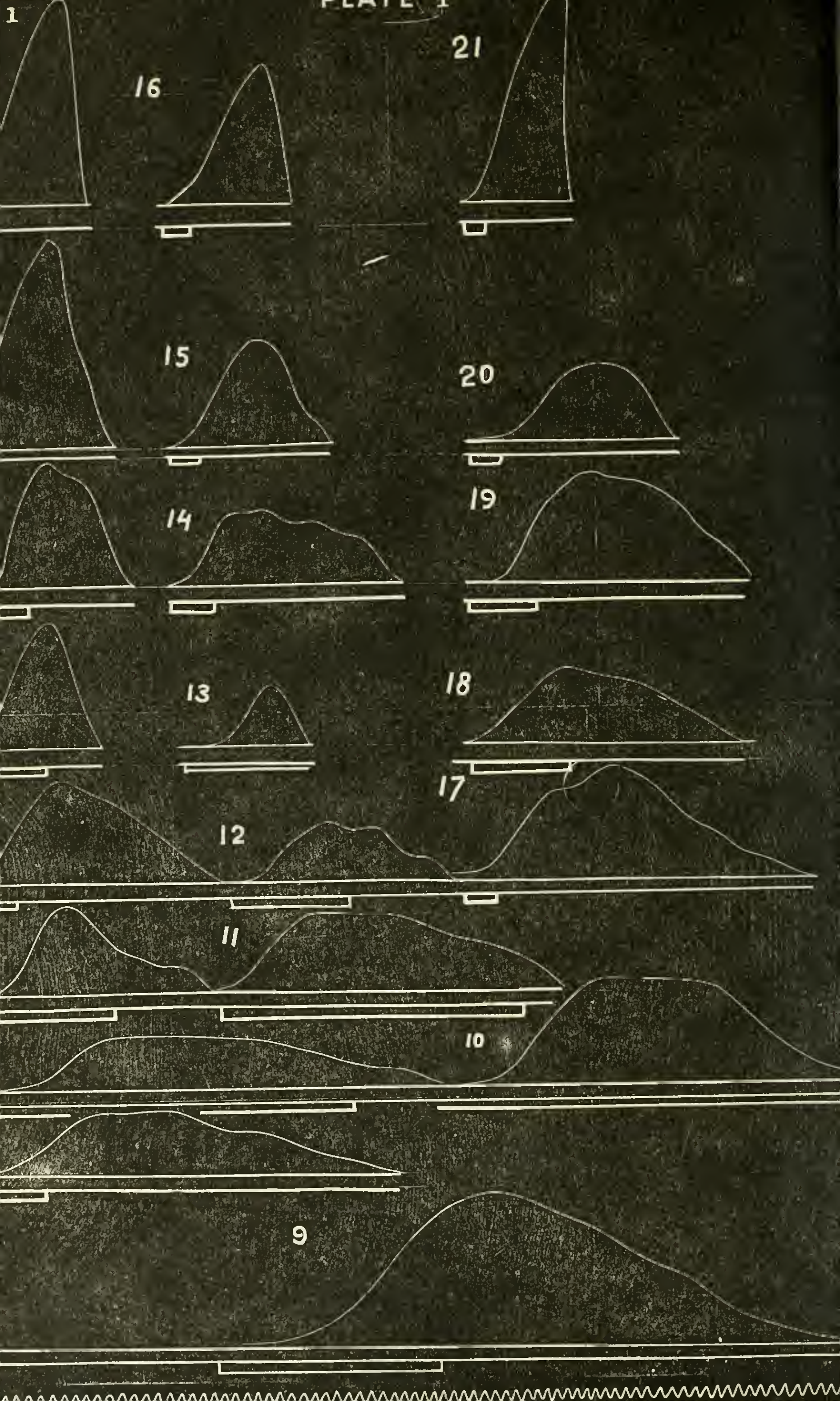

PLATE i.—Human myograms from direct muscular excitation, reduced one third. Tuning-fork line showing $\frac{1}{66}$ths of a second.

FIG. 1.—Cacc 47 cells ; fig. 16, ancc 47 cells ; fig. 21, fc + 2, of normal abductor indicis muscle.

FIG. 2.—Cacc 42 cells ; fig. 15, ancc 47 cells ; fig. 20, fc + 2, of abductor indicis muscle in a case of long disuse of hand from rheumatism.

FIG. 3.—Cacc 28 cells ; fig. 14, ancc 40 cells ; fig. 19, fc + 4, of anterior tibial muscles in a case of progressive muscular atrophy.

FIG 4.—Cacc 35 cells ; fig. 13, ancc 35 cells ; fig. 18, fc + 2.5, of abductor indicis in a case of partial recovery from myelitis of the anterior horns in the adult.

FIG. 5.—Cacc 30 cells ; fig. 12, ancc 30 cells ; fig. 17, fc + 2, of calf-muscles in a case of pseudo-hypertrophic paralysis.

FIG. 6.—Cacc 47 cells ; fig. 11, ancc 47 cells, of opponens pollicis in a case of old section of the median nerve.

FIG. 7.—Cacc 47 cells ; fig. 10, ancc 47 cells, of abductor indicis in a case of simple atrophy of the first interosseous space.

FIG. 8.—Cacc 30 cells ; fig. 9, ancc 25 cells, of the abductor indicis in a case of chronic myelitis of the anterior horns simulating commencing progressive muscular atrophy in the distribution of the wasting.

lepra anæsthetica with neuroma on each ulnar nerve, and with anæsthesia and atrophy in their distribution. The ancc curve (5) is identical with the tetanoid curves compared on the page 16.

There are some changes which muscles undergo by which their electrical reactions are changed, but not much so, and hardly enough to be visible to the unaided eye. These changes are principally quantitative, not qualitative.

Let us see how much our graphic method will help us in this matter.

The immobility consequent on long disuse of a part, as by retention of a member on a splint for a fracture or some inflammatory trouble, as rheumatism, every one has seen and undoubtedly has thought it more than can be accounted for by the false anchylosis which is often present. Persons acquainted with the use of electricity have also noticed that the nerve and muscle reactions in these cases are slow, but not qualitatively changed. Myograms 2, 14, 15, pl. ii, and 2, 15, 20, pl. i, show the changes. The muscular contraction is delayed and slightly tetanized. All authorities agree that there is no true degeneration reaction in progressive muscular atrophy, but this fact is better demonstrated by consulting pl. ii, 3, 12, 13, and pl. i, 3, 14, 19.

In pseudo-hypertrophic paralysis the normal formula is retained, but the myograms are peculiar, pl. i, 5, 12, 17, and pl. ii, 4, 10, 11. A word before closing as to the reactions in a peculiar case of paraplegia occurring in a girl who had recovered from a specific paralysis of the sixth and seventh nerve on the same side. The paraplegia was simple (no bladder or rectal trouble), and was almost confined to paresis of the anterior tibial and peroneal muscles. The reactions are interesting because they resemble degeneration reaction to galvanism as put down in the books, but faradic contractility is still retained.

The traces were made by excitation of the peroneal nerve and the anterior tibial group of muscles.

The nerve reactions were:

$$\left\{\begin{matrix}\text{anoc 15 cells,}\\ \text{pl. v, fig. 13,}\end{matrix}\right\} > \left\{\begin{matrix}\text{ancc 15 cells,}\\ \text{fig. 2,}\end{matrix}\right\} > \left\{\begin{matrix}\text{caoc 20 cells,}\\ \text{fig. 14,}\end{matrix}\right\} > \left\{\begin{matrix}\text{cacc 20 cells,}\\ \text{fig. 1;}\end{matrix}\right\}$$

an almost complete reversal of the formula.

The muscle reactions were:

$$\left\{\begin{matrix}\text{caoc 10 cells,}\\ \text{pl. v, fig. 11,}\end{matrix}\right\} \text{ almost } = \left\{\begin{matrix}\text{cacc 10 cells,}\\ \text{fig. 3,}\end{matrix}\right\} > \left\{\begin{matrix}\text{ancc 21}\\ \text{cells, fig. 4,}\end{matrix}\right\} > \left\{\begin{matrix}\text{anoc 30 cells,}\\ \text{fig. 9.}\end{matrix}\right\}$$

There was delayed and slow fc in nerve and muscle, which of course precluded the possibility of a destruction of the anterior horns of the spinal cord, as the paraplegia was of five months' standing.

A careful perusal of the explanatory text accompanying the plates and a comparison of the different traces among them will repay the reader by noticing:

First, the identity as to form and relation of human and frog myograms in health.

Secondly, the great similarity of the pathological myograms in frogs and man (section or disease of nerves).

Thirdly, the marked myographic changes where slight trophic disturbances affect human muscles (disuse, muscular atrophy, etc.).

Fourthly, the profound alterations in contour of myograms of muscles for a long time severed from the vitalizing power of their nerve centres (chr. musc. atrophy, chr. poliomyelitis, etc.).

The future of human myography cannot be predicted. It may become useful as a very accurate mode of differential diagnosis and prognosis. As an adjunct to our labora-

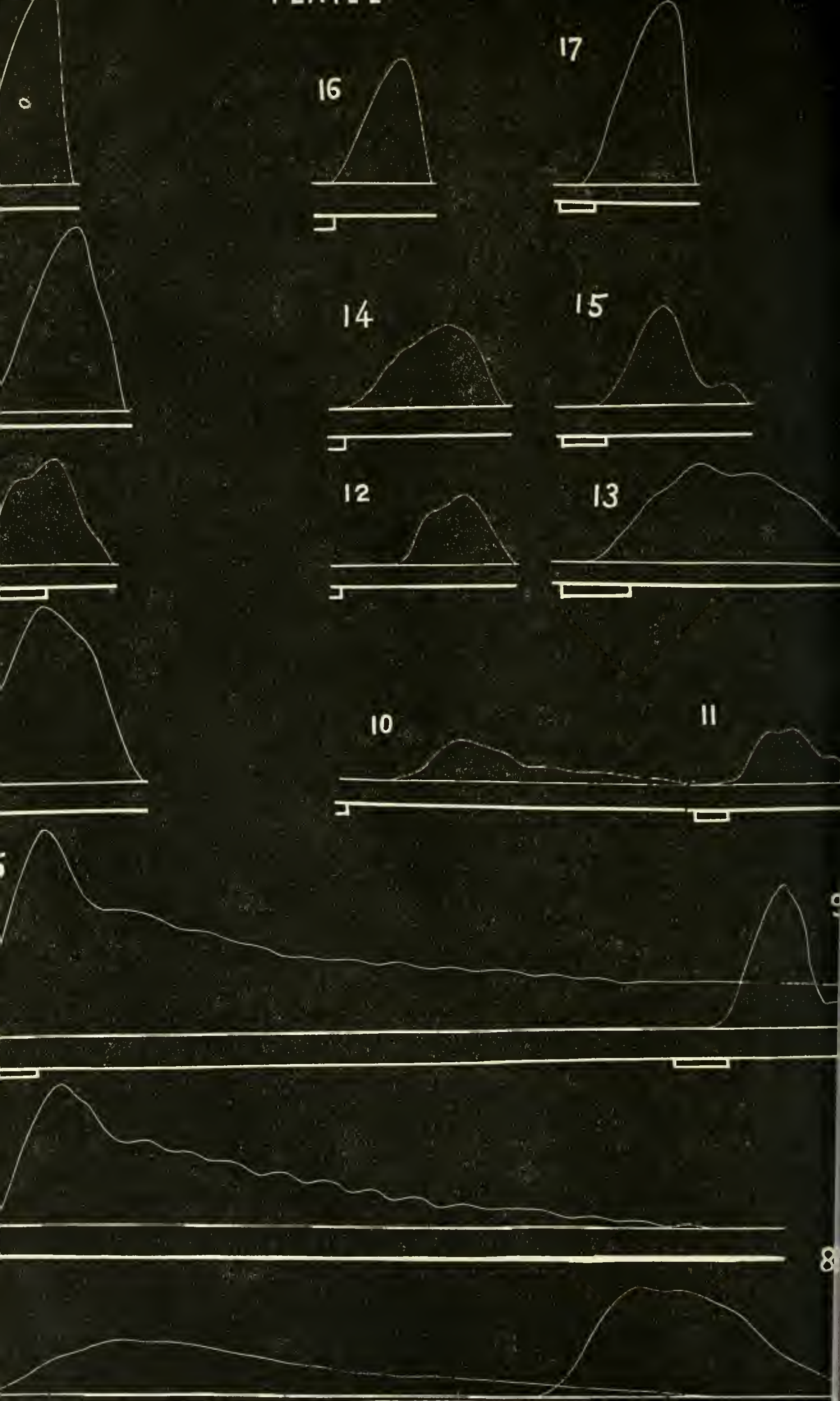
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PLATE ii.—Human myograms, all but last two produced by excitation of nerves ; reduced one third. Tuning-fork mark at the bottom = $\frac{1}{66}$ second.

FIG. 1.—Contraction of normal abductor indicis, cacc 32 cells ; 16 = anoc 40 cells ; 17 = fc contact.*

FIG. 2.—Case of long disuse of hand from rheumatism and splint ; abductor indicis. 2 = cacc 27 cells ; 14 = anoc 42 cells ; 15 = fc + 2.5.

FIG. 3.—Case of progressive muscular atrophy. Myograms of the anterior tibial muscles. 3 = cacc 28 cells ; 12 = anoc 34 cells ; 13 = fc + 4.

FIG. 4.—Case of pseudo-hypertrophic paralysis. Myograms of calf-muscles. 4 = cacc 25 cells ; 10 = anoc 25 cells ; 11 = fc + 2.

FIG. 5.—Old hemiplegia with contracture. Myograms of abductor indicis. 5 = caoc 35 cells ; 6 = anoc 40 cells ; 9 = fc. + 5.

FIG. 7.—Case of atrophy of first interosseonous space, not very progressive. Degeneration reaction. Myograms of abductor indicis. 7 = cacc 47 cells ; 8 = ancc res. fc. These last two traces were taken by direct excitation of the muscle, there being no nerve reaction.

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* Here and elsewhere contact means Dubois' secondary coil touching the primary. + 2 means the secondary coil overlapping the primary 2 centimetres. — 2 means the secondary coil being distant from primary, 2 centimetres.

tories for the study of experimental therapeutics it needs no lauding. Its employment is far more simple and occupies much less time than one would think, and the American apparatus is so much cheaper than the imported, that it comes within the reach of almost any one.

PLATE V.—Pathological human myograms. Nerve reactions in case of specific paraplegia, etc.

FIG. 1.—Cacc 20 cells; fig. 14, caoc 20 cells.

FIG. 2.—Ancc 15 cells; fig. 12, fc; fig. 13, anoc 15 cells, same case.

FIG. 3.—Cacc 10 cells; fig. 11, caoc, 11 cells, same case; from muscular excitation.

FIG. 4.—Ancc 21 cells; fig. 9, caoc, 30 cells; fig. 10, fc, same case; from muscular excitation.

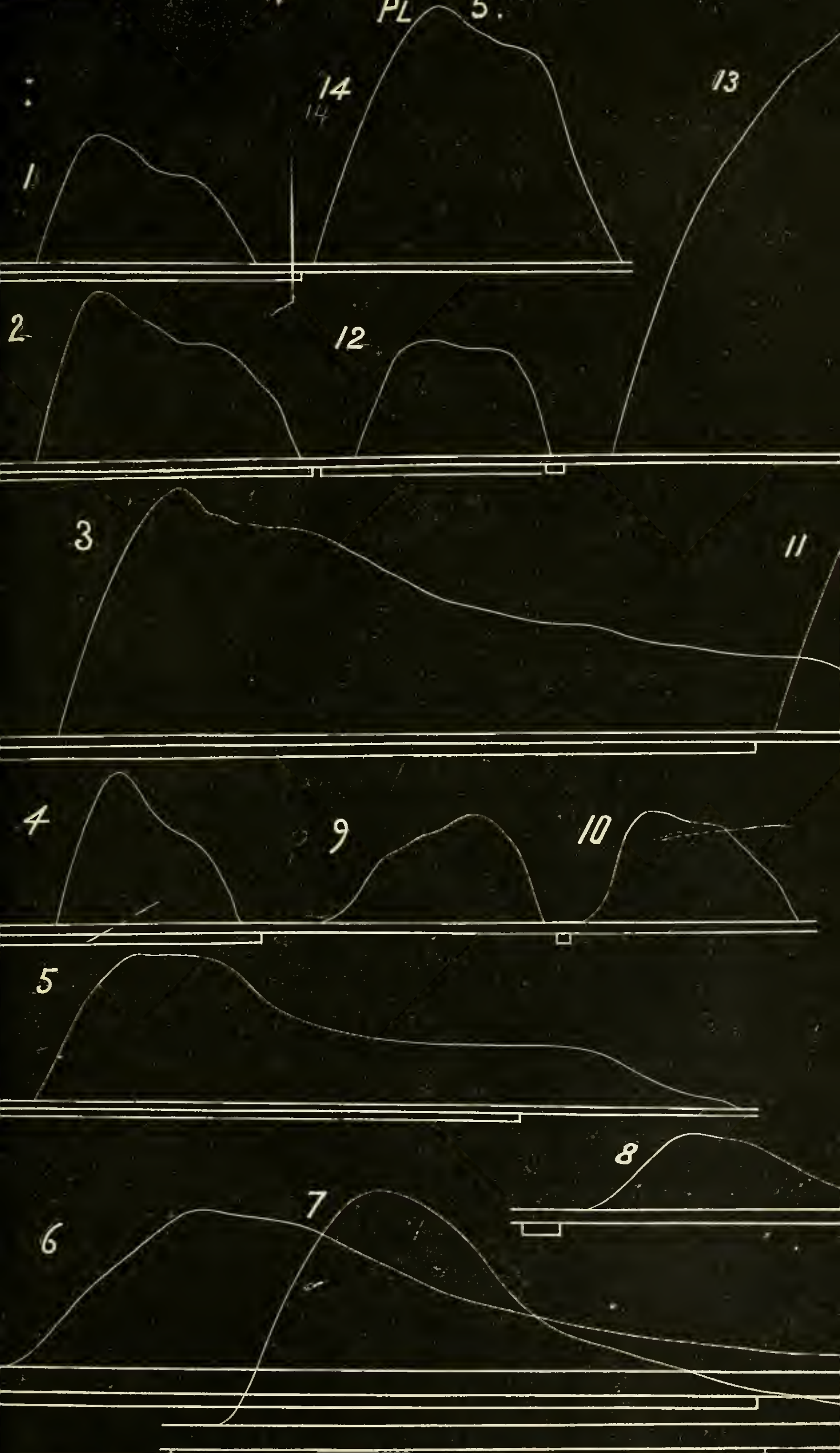
FIG. 5.—Ancc 35 cells; and fig. 8, cacc 47 cells, in case of lepra anæsthetica. Muscular excitation.

All the above curves reduced one third.

FIG. 6.—Cacc 47 cells; fig. 7, ancc 47 cells, in case of functional atrophy of the hand by direct excitation of the abductor indicis muscle.

NOTE.—For all the curves except 6 and 7, the tuning-fork line = $\frac{1}{66}$ths of a second; for curves 6 and 7 it denotes $\frac{1}{100}$ths of a second.

PL 5.



LEAD PARALYSIS.*

By S. G. WEBBER, M.D.,

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MY object in presenting a few cases of lead paralysis is not to discuss all the questions which naturally occur to one in connection with the subject. I shall limit myself to a brief review of the theories of its pathogenesis, and secondly to calling attention to some unusual forms in which lead poisoning may show itself, simulating more serious lesions.

There are two views, now most prevalent, in regard to the seat of the lesion in lead paralysis. Some authors consider the central nervous system, the spinal cord and brain, to be the parts primarily affected. Erb, E. Remak, Eulenberg, Monakow, Bernhardt, de Watteville, favor this theory; Westphal, Leyden, Zunker, Gombault, Charcot, favor another view—that the primary seat of disease is in the nerves.

The earliest case, with autopsy and examination of the cord, of which I have found report is one by Lancereaux. (*Gaz. méd. de Paris*, 1862, p. 709.)

The patient was 37 years old, had suffered from lead colic at 14 years of age, had been paralyzed in forearms and legs for 10 years before death. At the autopsy the brain and medulla oblongata were found normal. Just above the cervical enlargement at the level of the second and third cervical vertebræ the spinal pia mater was discolored over an extent of four or five centimetres. Here the cord was small and seemed atrophied and slightly softened. Some of the motor nerve roots at this level were smaller than those either above or below. Just above the lumbar enlargement was a similar spot of softening, but less marked, and some of the anterior and posterior roots seemed atrophied. On right side only two nerve roots in the upper part of the brachial enlargement were atrophied, and in these were healthy nerve fibres mixed with some which were altered, showing fine fat granules in the place of the medullary sheaths. The nerve trunks, going to the muscles, which had suffered most change, were much more diseased than the nerve roots, some of the fibres being reduced to mere connective tissue.

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* Read at the meeting of the American Neurological Association, June 23, 1882.

The cord presented what appeared to Lancereaux insignificant changes—only some cells were more granular than normal, deformed and atrophied.

The muscles showed degenerated fibres mixed with those less altered, retaining their striations.

In a second case which he reports, where the paralysis had been of four years' duration, the nerves supplying the affected muscles were much affected, their myeline being finely granular. The cord was normal in structure, but showed a congenital anomaly of development; the nerve cells, however, being healthy. Many of the fibres of the affected muscles were reduced to the sarcolemma with nuclei enclosed therein. (Lancereaux, Saturnisme chronique avec accès de goutte et arthrites uratiques. *Gaz. méd. de Paris*, 1871, p. 385.)

C. Friedländer (Anatomische Untersuch. eines Falles von Bleilähmung nebst Begründung der myopathischen Natur dieses Affection. *Virch. Arch.*, Bd. 75, 1879, p. 24) found the nerves degenerated within the muscles, also in the nerve trunks and even in their roots. There was no change in the spinal cord. The muscles had also undergone degeneration; the muscular nuclei remained.

Friedländer considered that the change begins in the muscles, and that the nerves are affected only secondarily.

Gombault (Contribution à histoire anatomique de l' atrophie musculaire saturnine. *Arch. de Physiol.*, v, 1873, p. 592) found the spinal cord and nerve roots healthy. The peripheral nerves were much altered. The nerve fibres were reduced in size; many contained granular myeline, and some sheaths were folded upon themselves, enclosing series of ovoid nuclei. The interstitial tissue was increased, and its nuclei were also multiplied in number. This was especially the case around the blood-vessels.

He comments upon: 1. The generalization of the lesion, spreading to a considerable number of muscles, after beginning in the posterior part of the forearm, where it reached its greatest intensity. 2. The so-to-say systematic distribution of the lesion in the muscles, explained by the neuritis of corresponding nerve trunks. 3. The perfect symmetry in the absence of any alteration of the central nervous system. 4. The nature of the process, which consists essentially in a proliferation of the interstitial tissue, while the proper elements of the organ degenerate and are atrophied. He refers to a similar change in the kidneys.

F. Tiburtius (Die Extensorenlähmung bei chronischer Bleivergiftung. *Inaug. Dis.*, Zurich, 1876. Only quotations from this have been seen) found the most important changes in the intramuscular fibres, which were greatly atrophied. The same change, less intense, was found in the musculo-spiral nerve, which was surrounded by connective tissue rich in nuclei; the interstitial connective tissue was not increased. The spinal cord and brain were normal. The patient had the usual form of paralysis, and also suffered from epilepsy.

Eisenlohr (Idiopath. subacute Muskellähmung und Atrophie. *Cbl. f. Nervenheilk.*, 1879, p. 100) found the muscles much changed; also marked atrophic change in the intramuscular nerves and the nerve root, the roots less than the muscular nerves. The cord was completely intact.

C. Westphal (Ueber eine Veränderung des Nervus radialis bei Bleilähmung. *Arch. f. Psych.*, etc., iv, 1874, p. 776) reports in regard to the changes found in Bernhardt's patient. The radial nerve showed a diminution in the number of normal nerve fibres without increase of connective tissue. The place of the nerve fibres, which had disappeared, was occupied by groups of small fibres without axis-cylinders. He considered these to be nerve fibres in process of regeneration. In the cord and nerve roots he found no change from the normal structure. Subsequently (vol. vi, 1876, p. 802) he says he thinks the above condition of the nerve fibres is less proof of the pathological change than when he examined the nerve, as he found a similar condition in normal nerves, but never in such excess as in the lead case.

Charcot (*Compt. rend. de la Soc. de Biol.*, 1879, p. 46) says he has several times examined the spinal cord in cases of lead paralysis; has never found clear lesions as are found in spinal amyotrophy.

Déjérine (Recherches sur les lésions du système nerveux dans la paralysie saturnine. *Mem. de la Soc. de Biol.*, 1879, p. 11) examined the nerves and muscles in five cases of lead paralysis. He found the muscular nerves altered in all five; he also examined the radial nerves in different heights, and found the change in that nerve; in one case he could examine the radial at the point where it took its origin from the brachial plexus, and recognized the change there. He then examined all the anterior roots of the cervical region in four cases, and only some of the roots in the fifth case. In two cases the anterior roots presented changes similar to those found in the peripheral nerves, excepting that it was less intense; the nerve fibres were changed, and in some cases the axis-cylinders had disappeared; the connective tissue was increased. This hyperplasia of connective tissue was not regular in its distribution. In the other three cases there was no such change discoverable in the nerve roots.

Déjérine does not give with this memoir the examination of the spinal cords, but rather promises that at some future time. In the discussion which followed his paper (*Compt. Rend. de la Soc. de Biol.*, 1879, p. 46), he said that he had not found any characteristic lesions in the cord, only some pigmented cells.

Although his examination, as given, includes only the nerves, he does not hesitate to state his belief that the primary lesion is to be sought in the spinal cord.

In six cases of which we have full reports the spinal cord was found healthy; Charcot, besides, says in a less definite way that he has several times found the cord healthy. In Lancereaux's first case, the cord was somewhat diseased, but apparantly not enough so in his opinion to cause all the disturbance in the muscles and nerves. In all the above cases the nerves which supplied the affected muscles were diseased, and in some of these the proximal parts are said to have been less affected than the more distal. Déjérine does not give his examination of the spinal cords in his five cases in detail, but says in a general way that he had not found any characteristic lesions in the cord; and though this was said in the discussion which followed the reading of his paper, it would not be unreasonable to believe that he referred to his five cases. He found, like the others, the nerves near or within the muscles most diseased; the nerve roots were not diseased in three of his cases, and only slightly diseased in two.

I. Moritz (*Journal of Anatomy and Physiology*, Oct., 1880, p. 78. A contribution to the pathological anatomy of lead paralysis) was unfortunately able to examine only the nerves and muscles; he could not examine the nerve roots nor the spinal cord. The intramuscular nerve fibres showed considerable thickening of connective tissue and multiplication of nuclei; in some fibres the axis-cylinders could no longer be seen. "The intramuscular nerve fibres were more uniformly and more extensively affected" than the muscles, "and hence it is probable that the process commenced in them and that the myotic changes were merely secondary."

The value of this observation is slight, but as far as it goes it favors the view that the nerves are primarily affected.

The evidence obtained by these observers from *post-mortem* examinations would be conclusive that lead paralysis does not depend upon lesion of the cord; but objection has been raised that it requires only a very limited lesion in the cord to give rise to the symptoms observed, and it is said that the examinations have not been complete. Remak (Zur Pathogenese der Bleilähmungen, *Arch. f. Psych. u. Nervenk.*, vi. 1876, p. 1) located the origin of the nerves supplying the extensors of the forearm above the cervical enlargement, and suggested that the examination had not been directed to that portion of the cord. He believes the lesion is seated in the cord, chiefly because one group of muscles which naturally act together are affected to the exclusion usually of another muscle, the supinator, which, also supplied by the radial nerve, acts rather with the biceps, and is probably supplied by fibres coming from another level of the cord. He also derives argument for his view from other forms of paralysis of spinal origin, as infantile paralysis, where groups of muscles acting together are affected to the exclusion of other muscles lying near but not acting with them.

Three years later, in the same journal (vol. 9, 1879, p. 572), he withdrew part of what he said as to the origin of the nerves going to the extensors, and said it is more probable that the nuclear origin of these nerves lies in the cervical enlargement itself, and, indeed, near its middle section, and here is where the anatomical changes in ordinary forms of lead paralysis are to be sought (p. 574). This is the locality which has been carefully examined by several observers and nothing abnormal found.

Four observers have found the cord diseased. One of these, Zunker (*Zeitschrf. kl. Med.*, i, 1880, p. 496), does not refer the symptoms to the cord as its primary seat, but thinks the nerve is first affected; that is, that the paralysis from lead poisoning is owing to a neuritis. He found the nerves in their smaller divisions greatly changed, and this change decreased upward, and was not found in the nerve roots near the cord,

He also found disease of the cord, an irregularly distributed atrophy of the gray substance; the large ganglion cells of the anterior cornua were diminished in number and size; in spots in the lower dorsal region there was complete disappearance of these cells.

Another, Vulpian ("Maladies de la Moelle," 1879, p. 158), considers that the primary change is in the cord, and that the paralysis in lead poisoning depends upon a myelitis. He says he has seen alterations in the spinal cord in a case of lead poisoning. Some nerve cells contained vitreous, colloid masses; others were undergoing atrophy; there was perhaps a little multiplication of nuclei. In this case the peripheral nerves and the muscles were altered. He also found islets of sclerosis in the roots of the nerves of the cervical enlargement.

In a dog poisoned with carbonate of lead he recognized a clearly marked myelitis. In several regions of the cord the nerve cells were undergoing destruction; the nerve fibres were altered.

Birdsall (A contribution to the pathological anatomy of lead paralysis. The *American Journal of Neurology and Psychiatry*, May, 1882, p. 176) found slight changes in the anterior cornua in the cervical region. The peripheral nerves were not examined, but no decided degeneration was found in the anterior roots. Birdsall refers also to an unpublished case of Seguin's where there had been partial recovery from lead paralysis; slight changes were found in the nerves and muscles, and there was a slight reduction in the size and number of the cells of the anterior cornua in the cervical region. Both these cases as given by Birdsall are simply of value as showing that changes occur in the cord, but they do not aid in deciding where the pathological process commences.

C. V. Monakow (Zur pathologischen Anatomie der Bleilähmung und der saturninen Encephalopathie. *Arch f. Psych. u. Nervenk.*, Bd. x, 1880, p. 495) reports a case which is of more than usual interest and is very significant. He found lesions in the brain, the cord, and the nerves. The location of the lesions was such that the changes in the cord could not be secondary to the changes found in the brain, and, as the distal ends of the nerves were more affected than the proximal portions, the roots were not diseased, it is not probable that the changes in the nerves depended upon those found in the spinal cord,

He says, in reviewing the changes found in the cord and nerves: "If the pathological changes in the extensors, the radial nerve, and the right anterior cornu are compared with each other, there can be no doubt that a close connection exists between them. The principal change in the spinal cord was in the vicinity of the exits of the sixth, seventh, and eighth cervical nerves, at just the points whence arise the roots of the radial nerve, innervating the extensors, and since there were no other symptoms of motor disturbance in the affected arm, one must consider this spot in the middle nucleus as connected with the paralysis of the extensors. But another question is: "Whence did the paralysis arise—from the muscles, the trunk of the radial, or from the spinal cord?" He concludes that the origin of the affection is to be sought in the spinal cord, though recognizing that there are difficulties in this view from the absence of changes in the radial nerve near the cord, and in the nerve roots.

Erb (Ein Fall von Bleilähmung, *Arch. f. Psych.*, etc., v, 1875, p. 445) reports a case without autopsy, and concludes that we must look upon lead paralysis as most probably central, or at least of neuropathic origin, although the central lesion cannot yet be proved by any examination which have as yet been made.

In the nervous system there have been found, then, changes in the motor centres in the brain (Monakow), atrophy of the ganglion cells, thickening of the walls of the vessels, and increase of the neuroglia; in the cord were found changes independent of all cerebral changes; either the brain not being diseased, or, as in Monakow's case, there being none of the usual secondary degenerations connecting the lesions in the brain with that in the cord. In the nerves there have been lesions which were at a distance from the cord, and which could not be seen to have any anatomical connection therewith. Either, then, these lesions of the motor tracks and centres are connected in ways not yet discovered, or the lesion originates independently in each of the three places.

The fact that it is the motor tract which is generally most affected, though not exclusively, would favor the view that there is a relation between the different centres of change.

We have as yet no proof that the higher centres, as brain or cord, can cause lesion of distant parts of the nervous system, as cord or nerves, without a continuous tract of degenerative tissue intervening. Such a relation may in the future be shown to exist, but at present it is not proven. On the other hand there are a few facts, as experiments on animals and the changes in tetanus and hydrophobia, which tend to show that the peripheral nerves may exert an injurious influence upon the nutrition of the cord, even when the intervening part of the nerve is apparently unaltered. In lead paralysis the mass of evidence derived from pathological anatomy would show that the disease is either primarily a neuritis, or the origin by separate and independent centres may be the true explanation. There are several considerations which would justify the latter conclusion. In Monakow's case and in some others there were changes in different parts of the nervous system which seemed to have no connection one with the other. Not only have there been found interstitial changes in the nervous system; the same kind of changes has been found in the kidneys (Gombault), and the bones have been found diseased (Lancereaux). The sympathetic ganglia have also been found affected with this interstitial degeneration. Probably, therefore, lead poisoning is favorable to the development of interstitial changes; when this influence is exerted upon the nervous system, we have symptoms which vary according to the seat of the change: cephalic symptoms when the brain is affected; spinal-paralysis, tremor, atrophy, according to location, when the disease is in the spinal-cord; and those which arise from neuritis when the nerves are affected; or a combination of symptoms when more than one portion suffers.

The fact that many cases of lead paralysis recover is in favor of the peripheral rather than central origin of the disease. Some years ago a worker in a white lead manufactory was under my care with extreme atrophy of the extensors of both arms; the muscles were, as it were, dissected out, leaving a cavity where they had been. There was entire loss of reaction to the faradic current, and the muscles responded very feebly to the galvanic current. After a long course of treatment he regained partial use of the muscles. Two years after, the muscles had recovered their normal size, responded perfectly to the will, and were of normal strength, only one muscle on the right gave a less than normally ready response to the faradic current. We cannot believe that the spinal cord is the primary source of the atrophy in this and similar cases. Most of the examinations of nerves and muscles in lead paralysis show that their nuclei still persist, long after paralysis has shown itself; from these nuclei may be developed the new structures which take their part finally in restoring the normal functions of the limbs. The fact that so many observers find the spinal cord intact may encourage us, then, to give a relatively favorable prognosis, unless the changes are so extensive and have lasted so long as to lead to the opinion that the cord is diseased. This, however, is a question in diagnosis which it will often be difficult to answer definitely, and we shall have to wait for time and the result of treatment to show how serious the changes have been.

Another fact, which has not been referred to as much as I think it merits, and which will lend weight to the view that the paralysis is caused by a variety of neuritis, is found in the sensory symptoms. In every case in which I have questioned the patients, there has been either pain more or less severe, or soreness, or a tingling sensation preceding the motor disturbance.

The first case which I report is one of undoubted lead paralysis; the girl was brought in contact with lead in her employment, and probably used white lead as a cosmetic; there was the lead line on her gums. The attack seems, from the patient's statement, to have developed in its last degree rather suddenly. Probably, if the patient had been more intelligent or less timid it would have been found that the loss of power in the legs was more gradual than the patient's account would indicate.

Case i.—Susan D., æt. 18. One year ago began to have pains in limbs; was sick three months, after which she was well till about a month ago; then she began to vomit, and had severe pain in stomach. Two weeks ago she lost the use of her legs and hands, and wrist dropped. Pain in the bowels has been less since the paralysis came on. A few days before the loss of motion there was a sense of numbness in soles of feet and legs, as if they were "asleep." The loss of power came on suddenly; she rose in night and could not stand; the next morning power of motion was gone. The loss of power in hands was more gradual.

In both arms paralysis of extensors and supinators; flexors and interossei still act. All the motions that are possible are executed very feebly and with considerable tremor. The faradic reaction is lost in the paralyzed muscles; retained, but diminished, in those over which she has partial voluntary control. Galvanic current, 25 cells move muscles on left; have no effect on right.

She could turn in bed, could draw legs up feebly, could perform some of the motions with feet, but feebly. Faradic reaction much diminished; 25 cells galvanic current move muscles of legs slightly.

Well-marked lead line on gums.

Urine contained no albumen; some hyaline casts.

Was treated with galvanism and iodide of potassium and iron. It was necessary to suspend the iodide occasionally on account of symptoms of acute lead poisoning, which arose during its use, but which subsided as soon as the medicine was omitted.

She stayed in the hospital six months, and when discharged was able to use her hands reasonably well; was able to walk fast; could not run.

Five months later she was in hospital again, still able to walk, right arm not quite so strong, troubled with colic. She improved, but would not stay more than about a month.

The second case was one in which syphilis might have been an element in the etiology, but lead being found in the urine it was thought more probable that metal was the cause of the paralysis. The recovery was more complete than I have generally seen in cases of syphilitic disease of the cord. Subsequently the man was in the hospital again for Bright's disease, during which plaques muqueuses appeared.

Case 2.—James H. S., æt. 28, was a hard drinker; complained, on entrance, of pain in abdomen, constipation, and weakness of legs. When seen, the patient was in a dull mental state, as if recovering from a severe drunk. The legs could only be moved feebly; there was entire absence of plantar reflex; the cremasteric reflex persisted. Patella tendon reflex was absent. Sensation was preserved to a limited extent, but his condition was such as to render accurate examination impossible. There was œdema of lumbar region and of the legs. The hands and arms could be moved naturally.

Expiration was prolonged over the bases of both backs, vocal resonance was more clear on left than right, percussion resonance equal on the two sides. Murmur with first sound of heart at apex, area of dulness slightly increased. There was tenderness over right hypochondrium; the hepatic dulness extended below the margin of the ribs.

A few days later he complained of numbness in his hands, and the strength was diminished in them, and finally he could not extend hand and fingers; could close hand feebly. The cremaster reflex was then absent, and there was priapism, the penis being constantly semi-erected.

Respiration was natural, but he complained of shortness of breath.

Urine was without albumen and casts; contained lead.

Pain in back led to the use of dry cups, which gave relief.

About seven weeks after entrance he began to regain freer motion of hands; the right, which had suffered less than the left, could be fully extended, the left nearly so; grasp was stronger. A month later he could stand and take a few steps without support. He finally recovered so as to be able to go back to his duties in the fire department.

The third case is of interest as showing the good result of persevering treatment; and while in the hospital it was interesting to compare her symptoms with those of another patient with myelitis, in whom the hands and legs were similarly affected, the muscles showing the same changes to electrical reaction, in whose urine no lead was discovered, who remained in the hospital nearly as long, but did not improve.

CASE 3.—Margaret F., æt. 27, married, entered Nov. 8, 1881; no children; one miscarriage three years ago. Her mother died from paralysis, and her father from the effects of a bad cold. Two years ago she lost the use of both lower extremities, and was troubled a good deal by excessive weakness of the arms. She remained in bed thirteen months; finally recovered completely.

About two weeks ago again began to have partial paralysis of legs and weakness of arms; the loss of power has increased. Preceding the commencement of this paralysis she had headache and dizziness for about a week, and during that week noticed that her knees seemed to give out on going down stairs; also had sharp pains in the calves of her legs; there was also some numbness in feet and hands.

She could move her legs slightly in bed; they were kept slightly flexed all the time; she could not extend them entirely on account of discomfort about the knees. Reflex from irritation of the sole of feet was slightly delayed. Sensation to touch in the legs was slightly diminished. The extensors of both hands were paralyzed, the flexors weak; there was no pain on passive motion of the hands; the opponens pollicis acted. Motions of the shoulder and elbow were fairly strong; there was a slight diminution of the sense of touch in the hands. The tongue protruded straight; the pupils reacted normally; there was no facial paralysis, no lead line on gums.

On the right the muscles of the forearm and hand responded to the faradic current; on the left the extensors did not act; the other muscles responded only feebly. In the legs the tibialis posticus acted feebly, the tibialis anticus not at all, to the faradic current. In the arms the galvanic reaction of the extensors was increased, as compared with the flexors.

Iodide of potassium was given, and after some days the urine examined for lead. A small amount of lead was found. Electricity, massage, and passive motions were used; the iodide of potassium was continued. After about two months she began to regain power in moving the hands; the fingers and hands could be partially extended; the right hand gained more and faster. Passive extension of legs and flexion of the feet caused considerable pain. She was discharged about the middle of March, 1882, able to walk with help. The faradic current did not cause contraction of any of the muscles of the legs, but the will had control over the muscles of the thigh, and she could move the toes; the affected muscles responded with more than normal readiness to the galvanic current.

The fourth case was rather unusual in its grouping of symptoms; in some respects resembling lateral sclerosis, there being stiffness, contraction, or rigidity of the limbs, and very greatly exaggerated patella tendon reflex.

Case 4.—Mary J., married, æt. 23, was well, excepting for last two or three years she had headache occasionally; three months ago began to have numbness in both feet and hands, gradually increasing, attended with rather constant pain running up knees and elbows; legs were swollen. For three weeks she was unable to walk on account of stiffness of legs. No cramps nor twitching of legs; they do not draw up in sleep; catamenia regular; other functions of body in normal action.

Both feet were entirely paralyzed, in extension; attempt to flex feet caused pain in feet and under the knees. Sensation was delayed in the feet four or five seconds; sensation of pain persisted after impinging body was removed. Above knees sensation was normal. Absence of plantar reflex. She could draw her legs up slowly and with considerable effort. Patella tendon reflex exaggerated.

In the upper extremities motion at the shoulders was moderately good; at the elbow very weak; extension of the wrist and fingers lost; flexion of wrist and fingers feeble; action of interossei nearly lost; rotation of arm feeble.

Gums were shrunken away from the teeth; there was no lead line.

The faradic reaction was diminished or lost in the muscles most affected. No separate note was kept of differences.

The urine was without albumen; contained considerable lead after iodide of potassium had been given.

Galvanic current and massage were used. Iodide of potassium was given internally.

She was under treatment nearly five months, during which she regained partial power over the hands, and recovered use of legs so that she could walk about without help.

Excepting the last case I could give the histories of other patients under treatment at the same time with very similar symptoms, in whose urine after using iodide of potassium no lead was found, and who did not improve under treatment. It would extend this paper to too great a length, and would probably add nothing to its practical value.

Dr. F. Minot published some cases of lead poisoning in the *Boston Med. and Surgical Journal* for March 10, 1881, in which there were symptoms resembling those of myelitis. I desire to emphasize that peculiarity in cases of lead poisoning. We have known all along that the legs may be paralyzed as well as the hands, but it is not so generally recognized or mentioned that lead poisoning may almost exactly imitate myelitis. The value of an examination of the urine for lead after administering iodide of potassium becomes, then, self-evident. The lead line was wanting in most of the cases reported, and is often absent in undoubted cases of lead poisoning.

In only one of the four cases could the origin of the lead be discovered. This is perhaps not strange in view of the results obtained by A. Gautier (*Gaz. Hebd.*, Nov. 11, 1881, p. 724), who examined different articles of food and found lead in preserved vegetables, fish, crustacea, meats, also in drinks and drinking water, and the water artificially charged with carbonic acid, in acid drinks and condiments put up in glass, in vessels and utensils of tin, in the glazing of the walls and furniture, the woolen and silk of our clothing, the leather of our shoes, in enamelled cloth. In the oil in which sardines are put up he found a relatively large proportion of lead.

SOME OF THE FACTS OF LISTERISM, WITH A COMMENTARY.*

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IN opening this discussion I shall first state what Listerians claim for the method, and give the results obtained, not from selected cases, but in *all* their operations, extending over prolonged periods of time. I shall then give my own experience of the method. I shall next explain the principle of Listerism, which seems to be misapprehended by many surgeons in this country, and in consequence, a fair trial of the method is rarely given, with the consequent result of an almost total disbelief in the positive statements of some of the most prominent of foreign surgeons who have become converts to aseptic surgery. The objections urged against the method will then be considered. Finally, I shall endeavor to show that this, as well as all other modern wound-dressings, is dependent for its success upon certain general scientific principles.

It will be of course impossible to more than lightly touch upon the salient points of my subject, but I shall endeavor to bring prominently forward all that is really essential for its comprehensive discussion.

Lister and his followers claim that by the recognition of certain general principles suppuration may be avoided, or at least reduced to a minimum; that at the present time certain procedures have been found to ensure this success better than others, but that as the method has been much modified in the past, it may very probably be simplified in the future; and very justly maintain that *the indications remaining the same*, the methods may vary without the essentials of Listerism being altered. They contend that aseptic surgery does not consist in the use of the spray, or any special dressing, but in complying as perfectly as possible with certain principles. It is not pretended that, under favoring circumstances, essentially similar results may not be obtained *exceptionally*, but it is asserted that, under the most unfavorable circumstances, where, previous to the introduction of Listerism, certain operations were almost invariably fatal, a dangerous mishap is as rare now as a recovery was before. The Lister school has been unfairly charged with making very absurd statements, but my last sentence really contains what they actually claim.

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* Read before the Phila. Academy of Surgery, June 4, 1882, to open a discussion on Antiseptic Surgery.

Let us now examine the results obtained in *all* the cases treated aseptically by various surgeons.

In his least healthy wards, Lister for four years suspended the annual washing. He overcrowded them, having several patients in *one* bed. The dressings were untouched from six to eight days, with but *one* case of pronounced pyæmia. In the same hospital he treated another series of operation-cases, etc., with the ordinary methods then in vogue. They amounted to several hundreds, and covered a sufficiently long period of time to eliminate any probable sources of error. As compared with his *aseptic* cases, he had nearly *double the mortality, and that from septic disease.*

Volkmann, of Halle, when he commenced Listerism, was in such despair on account of the unsanitary condition of his wards as to be on the point of demanding their closure. As an instance, in 1872–3, he tried to save—and they must, of course, have been comparatively simple ones—16 cases of compound fracture, and 12 rapidly died of pyæmia. Under Listerism, from 1873 to 1877, he tried conservative treatment in 75 complicated fractures in 73 patients, *without a death.* Let me give *all* his operations at his clinic, from March 1, 1874, to March 1, 1877:

AMPUTATIONS, SIMPLE.

	Cases.	Deaths.
Shoulder,	4	1 in 4 hours.
Arm,	14	——
Forearm,	23	——
Wrist,	3	——
Hip,	2	1 in 4 hours.
Thigh,	42	1 in 24 hours.
Leg,	25	1, erysipelas.
Parts Foot,	42	——
	155	3, shock; 1, erysipelas.

RESECTIONS, SIMPLE.

	Cases.	Deaths.
Shoulder,	7	——
Elbow,	2	——
Wrist,	2	——
Hip,	48	4 { 1 child, 9 mos. 1 child, 2½ yrs. 1 after 3 mos.
Knee,	21	1, tubercular meningitis.
Ankle,	5	——
	85	5 in all.

Four excisions in pyæmic cases, *all fatal.*

AMPUTATIONS, COMPLICATED.

	Cases.	Deaths.
Double,	9	2, of both thighs.
Serious multiple lesions,	6	6 in a few hours.
During septicæmia,	15	8

One case at 60 years, died of delirium tremens.

One tubercular case, died of pneumonia.

One shoulder, aborted, and died of puerperal fever. In these latter cicatrization was nearly complete.

SECONDARY AMPUTATIONS.

	Cases.	Deaths.
Thigh,	1	——
Knee,	4	——
Leg,	42	——
Arm,	6	——
Elbow,	5	——
Forearm,	15	——
	73	

Hydroceles incised,
45 cases, no deaths.

	Cases.	Deaths.	
Resections for false joints or vicious callus,	10 (9–1).	—	
Osteotomies,	50 in 38 cases.		1, in a bleeder.
Amputations of Breast,	119 in 115 cases.	6	2, from erysipelas: one arising from a neglected dressing, one from a bed-sore. 1, shock. 2, exhaustion (pyæmia?). 1, malignant pustule.

If dissatisfied with the statements of continental surgeons, examine the opinions expressed by such men as Spencer Wells, C. Macnamara, Barwell, T. Smith, J. Wood, Jonathan Hutchinson, Sir James Paget, Knowsley Thornton, etc., who, either in all operations or in certain of the more dangerous, extol Listerism in the highest terms.

Mr. Wood says: "I estimate highly the spray and gauze treatment in the opening of psoas, spinal, or other large abscesses for the first time."[1] J. Hutchinson says: "I have seen large abscesses opened under the spray and dressed with gauze, which did not suppurate further, and which behaved in a way wholly unexampled under the older methods of dressing. I have also seen many large operation-wounds so treated heal without suppuration, and without the slightest approach to a febrile condition." Paget says: "But there are some groups of cases in which I believe it would be absolutely wrong to dispense with any of the precautions of the complete antiseptic surgery. Among these are the cases of ovariotomy. * * * As with ovariotomy, and, I may add, all abdominal sections, so with osteotomy and the cutting into healthy joints. * * * These may now, with antiseptic precautions, be done with an almost complete safety."

"A few years ago I believed that I had never seen a patient recover after the opening of a lumbar or psoas abscess with a free incision. I could not remember one who had not died before the opened abscess healed. Of late years I have known such abscesses opened with complete impunity under antiseptic treatment, and there has seemed nothing but this treatment to account for the difference of the results."

I will now examine in detail the results of aseptic treatment in amputations, injuries of joints, compound fractures, etc.

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[1] He means when not *previously* opened by the knife.

Scrutinize Schede's statistics of septic and aseptic cases. Thus, of 377 amputations performed by Bruns, Bardelebem, and Billroth, there was a mortality of 29.18%, while of 321 amputations performed by Busch, Schede, Socin, and Volkmann there was a mortality of 4.4%.

Bardenheuer had no death during a year at the Cologne Infirmary, with the following operations involving bones.

41 amputations (17 of thigh) through bones.
10 " at joints (1 at hip).
53 excisions of joints (15 hip, 12 knee).
23 cases removal wedge-shaped pieces of bone.
5 operations for badly united fracture.
1 case trephining.

As to joint cases, Bryant has criticised the small number of healthy articulations opened by Lister, and that several were of the smaller joints; but Mr. Lister has frequently opened those affected with hydarthrosis, impending suppuration, joint abscesses, etc., in all 109 cases, with but three deaths, all from tubercular meningitis. Volkmann gives 31 wounds of joints and 2 incisions into joints, 33 in all, of knee, wrist, elbow, and foot, with no death. He also gives 21 comp. fractures entering joints treated aseptically.

Resection was resorted to { 2 primarily.
{ 5 secondarily.
Amputation was required—3 secondary amputations.
10 perfect recovery, with free motion.
1 ankylosis (not seen for 10 days).
—
21 No deaths.

In four cases late treatment or gangrene compelled operation.

Nussbaum says: "This method opens up a new field to surgeons. By the aid of these precautions, joints and cavities of the body may be opened without danger"; this, too, in an unhealthy hospital. Hear Heuter: "It is with reason that I said, in 1870, that puncture of joints ought to be considered as a dangerous operation; it is with as much reason that I now affirm (1876) that one can practise this operation without danger."

After omitting all cases of compound fracture dying within 48 hours, or which required primary amputation, taking *all* Mr. Lister's hospital cases from 1871 to 1881, there are left 39 fractures in 38 cases with only 2 deaths, one of which was independent of the wound; 31, or 81.5 % were rendered aseptic, and *all* of them recovered, viz.: 31 compound fractures of the skull and long bones. In 82 compound fractures made by Mr. Lister, nearly every case pursued an aseptic course, but in one of these amputation was required; *none died.* These operations were performed upon the femur 27 times; leg, 31 times; clavicle, 3 times; humerus, 9 times; forearm, 11 times; and lower jaw, once—82 operations in 58 cases. The favorable results are not due to the *youth* of the patients, for they averaged as such cases do. Besides, he has had some 22 other operations on bones of such severity as to be dangerous, without a single fatality.

MacEwen reports an extraordinary series of osteotomies. His operation-wounds varied in length from ¾ inch to 1½ inches, and were held apart so that the air had free access to the bones. He has operated on 330 patients, producing in them 885 compound fractures, but in only 8 did suppuration ensue. In only one instance was secondary amputation necessary, and here gangrene was produced by displaced bandages. Only 3 died out of 330 patients, one of whom had 10 compound fractures, the deaths being from diphtheria, tubercular meningitis, and pneumonia contracted before the operation. Most of his cases were also cachectic.

The accidental compound fractures of Lister, Volkmann, Schede, and MacCormac, in which an attempt was made to purify the wound, excluding all dying within 48 hours, amount to 164 cases, of which number only 2 died. Of 158 fractures of the extremities, secondary amputation was required in 15, secondary excision in 5, leaving 138 cures with useful limbs. The intentional compound fractures of Lister, Volkmann, MacEwen, Bardenheuer, and MacCormac, number 530 cases with 1,072 fractures, with but 3 deaths, all of which were independent of the operation itself. In 766 limbs 1,068 fractures were made, and but 4 cases required amputation. Again, adding both classes together, of 1,226 fractures of 924 limbs, 902 recovered, but 19 required secondary amputation, and 5 secondary excision. Of these, 5, or 0.72 %, died. Contrast this with the best results of compound fractures treated by the ordinary methods, viz.: 22 %, which were achieved at St. Thomas' Hospital. Finally, examine the results following free opening of psoas abcesses. By considering those lost sight of in Mr. Lister's report as dead, we have a mortality of only 9 in 37, or 24.3 %. Some of the deaths were also due to phthisis, etc. This operation—which requires the greatest skill and attention to details—is that which has failed oftenest in other than Lister's hands, yet as Paget, Hutchinson, and Wood have pointed out, success *is* attainable. Can any candid mind refuse to believe statements coming from so many and varied sources? Many more such records of facts could be adduced in support of my position, but if what has been already said does not carry conviction with it nothing will. I imagine that no one present has ever succeeded in performing 72 major secondary amputations without a death. The surgeons who have given these statistics are implicitly believed when publishing other than *Listerian* facts. Upon what grounds, then, have we the right to deny them credence where aseptic surgery is concerned?

I will not tire you with details of my own experience. My failures were many at first, my success was slow, but I am now confident that I can do still better. The traumatic fever was insignificant, the pain slight; the wounds remained *white-edged*, unswollen, and healed largely by first intention; the pus was insignificant and different from what was ordinarily seen; the wounds were perfectly sweet, even in hot August weather, after 7 days; when sloughing of flaps occurred the discharge was as nothing, comparatively, and *with no odor.*

My statistics although quite extensive are, in my opinion, too small to be of any such determinate value as to warrant publication, although convincing to myself of the value of Listerism. I may say that my best results were obtained when the wards were overcrowded with suppurating wounds to a degree which should have certainly, according to Erichsen, generated pyæmia, etc.

What is Listerism? It is the prevention of putrefaction in wounds by the destruction of particles which induce fermentation, and which Lister considers are micro-organisms. Lister recognizes three main causes of suppuration, which, when overlooked, as they seem to have been by many surgeons in their application of his method, ensure failure, viz.: (1) Excessive tension of tissues; (2) direct irritation of the living tissues by the presence of a foreign body; and (3) direct irritation by germs which are usually suspended in the air. The omission of attention to the first two causes has usually resulted in disappointment, and in consequence the value of Listerism has been denied. A single tight stitch may, and failure to provide the most perfect outflow for the serous discharges, which are peculiarly large from the irritant effect of carbolic acid, certainly will, *ensure* suppuration, although it may be aseptic. Again, irritation of the wound by deluging it at the outset and at each dressing with carbolic acid solution is almost *certain* to initiate the second cause of failure. The removal of the third cause of suppuration—and here is the point which most anti-Listerians overlook and then maintain that other methods besides the spray result in equally good results, *ergo* Listerism is false—may be effected either by the purification of the air, with consequent destruction of germs *before* they reach the wound; or, allowing them *free access* to the cut surfaces, they may be afterward *destroyed by germicidal applications*, and subsequently kept out by appropriate dressings. What I have just described is Listerism, or the principles of aseptic surgery. The *Listerian method* consists at present of spray, gauze, etc., but this is only one method of application of the Listerian principle, which, as it has varied in the past, may do so in the future. The mistake so commonly made is, that the spray, gauze, etc., constitute Listerism, *i. e.*, a certain *principle* of wound-treatment; forgetting entirely that Lister used none of these things in the past and yet got good results, that Trendelenberg uses no spray yet is a strict aseptic surgeon, etc., etc. When a man dispenses with the spray the cry goes up he has abandoned Listerism. He *has* abandoned the present Listerian *method*, but *not* the principle in most instances. Lister himself merely considers the spray as a *convenience*, which ensures results easily, and obviates the necessity for rapidity in operating and the deluging of wounds with an irritant like carbolic acid.[1] The most essential part of the theory is the thorough purification of every thing—instruments, hands, finger nails—which comes in contact with the wound, *and keeping them so throughout the operation.* The introduction of one infected instrument into the wound is vastly worse than the momentary deflection or cessation of the spray. If Lister had to give up any part of his *method* (mind, not principle) he would abandon the spray, because it is both theoretically and practically possible to render a wound aseptic without its aid, although we can have no *certainty* of attaining this end. From this we can see how many wound-treatments are practically aseptic, and why results analogous to Lister's are obtained by surgeons who ridicule, while in reality profiting, wittingly or unwittingly, by, not his *method*, but his *principles*.

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[1] Lister has pointed out that with good hygienic surroundings the air probably contains very few germs, certainly *none* compared to those lodged upon instruments, adhering to the hands, etc.

The chief objection urged against Listerism is that equally good results are attainable by means of cleanliness and hygiene. To this I would merely oppose the various facts that I have given, and especially that Nussbaum, with open method, disinfectants, etc., could get no such results until he instituted strict aseptic surgery, and that where this is impossible, he still has his *old accidents*. As von Buhl makes all the *post-mortem* examinations, and many practitioners and students see the cases, error can hardly arise.

Other objections from improper applications of Listerism I shall presently discuss. Another complaint is that carbolic acid is a poison. True, but almost never, and then chiefly when used antiseptically, not aseptically; *i.e.*, deluging the wound with quantities of the lotion. But Listerism does not consist in using carbolic acid. Salicylic acid, oil of eucalyptus, and acetate of alumina[1] are nearly as good. In passing, I would say, as a proof of the aseptic method, Volkmann tried thymol instead of carbolic acid, when he rapidly began to get bad results from the use of this inefficient antiseptic, although the same cleanliness, etc., was observed. Again, expense is urged, but this is hardly true, as fewer dressings, greater rapidity of cure, the rare need for tonics, febrifuges, etc., etc., and the shorter stay in hospital, render it in the end no more expensive. Finally, it is said to be troublesome. After a little practice it is not more troublesome than other methods—and if it were, no right-minded man could seriously urge such an objection if the results are as they are stated to be.

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[1] I have used this with quite as good success as carbolic acid.

There is one objection which is so illogical that I need merely mention it, viz.: that organisms are found under the dressings, *ergo* Listerism is a failure; or, in other words, the reasons assigned for success are untrue, therefore the success, which is demonstrable, is a delusion.[1] Did the earth cease to go round the sun because the older philosophers taught the reverse?

As time would fail for a thorough consideration of the subject, I will merely state a few bald facts as to the germ theory of putrefaction, referring my hearers to Cheyne, Pasteur, and others for details. One misapprehension must first be cleared up. Many surgeons confuse the germ theory of putrefaction with the germ theory of infective diseases, considering that they stand or fall together. This is a mistake, as there is no such necessary connection.

Fresh air frequently renewed, *i. e.*, oxygen, is more unfavorable to the growth of organisms than any dressing which imperfectly excludes the air. Indeed, Pasteur considers oxygen to be fatal to the development of the *vibrio septica*. In other words, the open method is better than any form of closed dressing, except Lister's, in preventing the growth of organisms. Concentration of wound-fluids interferes with their development, thus explaining, in part, both healing under a scab, Guérin's cotton-dressing, and the open method. All forms of germs are not equally harmful, as micrococci seem *not* to favor putrefaction, but bacteria do. The growth of some organisms is inimical to others. Bacteria do not develop from micrococci; these act differently with chemical agents, and have been seen to divide *longitudinally*, while bacteria do not. Bacteria flourish in alkaline fluids. Micrococci flourish in a proportion of carbolic acid which almost totally prevents the development of bacteria.

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[1] Stimson's experiments as to the efficacy of the spray are so manifestly faulty as to demand no refutation. No one thinks of *sweeping a room so as to raise a cloud of dust* while operating!

Contact with healthy tissues destroys micro-organisms unless they are present in large numbers. In acute inflammatory affections or disease, organisms may gain access to the economy by other routes than by wounds. Organisms are always absent from chronic abcesses, but are equally constantly found in acute abcesses. If the latter are not opened early, these organisms are dead, since they soon exhaust their nutriment; they can be seen microscopically, but fail to develop when cultivated. Carbolic acid, unless much concentrated, does not kill, but *anæsthetizes* the germs and they cease to develop.

Finally, as to the presence of micro-organisms in wounds treated aseptically. When the method has been properly carried out they do not appear until late in the treatment of the case, when the *dressings have been left on for a long time*, *i. e.*, the carbolic acid is, to a great extent, washed away or has evaporated. They appear first at the edges of the dressings, and travel from *without inward.* Where the wound remains aseptic *nothing* but *micrococci* are observed. Now note three things, viz.: micrococci are found in acute abcesses, the pus of which is *sweet* when opened; when in small numbers they, as well as bacteria, are killed by contact with healthy *uninflamed tissues;* carbolic acid, like chloroform, will anæsthetize them, as it will the yeast plant, so that their destruction is not absolutely essential; Onimus and Dumontpallier showed long ago that minute proportions of various antiseptics destroyed the septicity, as proved by inoculation, of previously septic blood, without apparently affecting the *vitality* of the micro-organisms. The bearings of these facts upon the explanations of the success of the open, modified aseptic, and aseptic methods of wound-treatment are too manifest to need comment.

Examine with me the pathology of wounds, and its bearing on the reasons for the success of certain plans of treatment, bearing in mind the just-mentioned facts regarding micro-organisms. For those that repudiate the germ theory of putrefaction, I need only say that the measures which have proved most efficacious in excluding organisms from wounds have also excluded the causes of putrefaction, and that, as I hope to show, the results can be satisfactorily explained upon another basis, although I am inclined to the germ theory myself. I shall now quote extensively from a paper published by me just a year ago.[1]

> "Let us first examine the condition of an amputation-wound after it has been put up. The capillary and smaller vessels are sealed by clots which extend to the next collateral branches; but, of course, these same vessels, where not filled with hæmostatic plugs, are in an excellent condition to absorb any thing brought into sufficiently close proximity. In a few hours, especially in women and children, where, owing to the thinness of the epidermis, the congestion is not concealed, a marked flush is seen. This increased vascularity is, of course, present in the deeper structures also. In the normal course of events this congestion is relieved by a more or less copious flow of serum, which is finally substituted by pus. As early as the third day—and I think that I have observed this change even before in quite a considerable number of cases,—by the aid of a lens, granulations may be seen springing up in those portions of the wound where perfect coaptation has been impossible.
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> "Let me recount one more set of preliminary facts experimentally proved by Billroth, viz., that an artificially prepared septic fluid can be placed in intimate contact with a *perfectly healthy granulating surface with impunity;* while another portion of the same, when injected into the cellular

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[1] "An Analysis of the Principles on which Depends the Success of the Modern Methods of Treating Wounds," by C. B. Nancrede, M.D., ARCHIVES OF MEDICINE, June, 1881.

tissue, will rapidly destroy the animal, with the most marked septic symptoms.[1] He also states that since in compound fractures he has adopted the use of the plaster-dressing, the occurrence of septicæmia and pyæmia has been much less frequent in his practice.[2] By the more perfect quietude insured by the fixed dressing, the granulations are not mechanically injured by the frequent, although slight movements of the fragments; the muscles are maintained in a state of quietude, and consequently the pus cannot be either worked into the cellular interspaces, nor into the granulations, or breaches in them produced by movements of the fragments and injudicious handling. Now what is the most natural inference from this? Plainly, that which experience has already taught us, that perfectly healthy, well-formed granulations are an efficient barrier to the absorption of septic materials, at least for a time. I do not deny that acrid septic fluids, whether produced by the addition of a special ferment from without, or generated by bad hygienic surroundings, systemic conditions, etc., may, like caustic, rapidly destroy healthy granulations and thus allow of poisoning of the system, but I am prepared to maintain that for a considerable time they do prevent absorption. Besides, I have been so very often told by those who should know better, that a given wound was doing beautifully with a perfectly healthy granulating surface, with the qualifying statement, however, that it refused to cicatrize as it should, and upon inspection have found that portion only of the professional opinion concerning the 'not healing' was correct, that I am always sceptical when I hear that a thoroughly healthy surface in all its parts immediately preceded septic trouble. It should also be borne in mind that all structures do not form granulations with equal rapidity, so that while the accessible portions of a wound may be to the eye healthy, other deeper parts may be in an exactly opposite condition.[3]

"Now let us examine the conditions of the wound, during the first forty-eight hours, as to sepsis. Whether we believe that suppuration—especially unhealthy suppuration—and septic trouble are produced by the importation of germs from without or not, there can be no question that the secretions of a septic wound, when applied to solutions of continuity in other individuals, will produce a similar constitutional affection. What are the conditions most favorable to such absorption? Clearly the presence of a secreting surface capable of absorbing, and a favorable nidus for the development of the *materies morbi*, whether it be chemical or bacterial.

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[1] Recent experiments upon this subject have given *apparently* other results; but they are clearly explainable in accordance with my statement.

[2] Viewed in the light of the above-mentioned experiment, the explanation of such good results is readily discernible.

[3] A careful analysis of the course of the majority of cases of blood-poisoning in all its forms, will demonstrate that the commencement of the trouble, *although unnoticed*, dates back to the first three or four days after operation or injury—indeed usually within the first forty-eight hours some *slight* symptoms appear, the gravity of which time demonstrates.

"The study of the physical conditions favoring osmosis shows us that an over-full condition of the vascular system, by the comparative stagnation of the contained blood, retards, if it does not entirely prevent absorption. This distended condition of the vessels I have already said soon supervenes after operation, and in accordance with this fact there is a variable but distinct period before any thing like marked fever shows itself. Soon, however, the overloaded vessels relieve themselves by a transudation of serum, or, perhaps more correctly, altered liquor sanguinis. This relative cessation of congestion acts in two unfavorable ways, however salutory it may in other respects prove, for it provides an abundance of a readily putrescible fluid well adapted for the propagation and development of bacteria and minute organisms, while at the same time it unloads the circulation to such an extent as, in the highest degree, to favor absorption. The normal vascularity of any part subjected to amputation is scarcely fitted for rapid absorption. The excessive primary vascularity just spoken of is still less favorable to osmosis, but the state of the blood-vessels, after their partial depletion, is such as to meet all the requirements for septic or other absorption. Soon, as we have seen, granulations commence to form, so that the risk of absorption is measurably diminished by the end of forty-eight to seventy-two hours. I would beg my readers to bear this fact in mind, as it gives the key, I believe, for explaining the success of von Brun's method.

"After these preliminary explanations, let us now—bearing clearly in mind the wound-conditions just described—examine what must be the indications to fulfil to avoid the risk of sepsis. Of course, no one doubts that if every wound could be made to unite by 'first intention,' or immediate union, the chances of septic infection would be almost, if not absolutely, done away with. To this end, the most accurate coaptation of the wound-surfaces, with their subsequent retention, is the primary essential. The most difficult portions to appose, and keep so placed, are the deeper parts, and the only agent likely to disturb the position of the flaps, after coaptation has been secured, is the accumulation of the secretions, due to some obstacle presented to their escape. Manifestly, then, the second indication is to secure free drainage. The third indication is clearly to remove inflammatory products as soon as secreted, so as to render impossible any accumulation of that putrescible fluid poured out from the cut surfaces, which affords such an admirable nidus for the development of septic poison.[1] This removal is of paramount importance, as is clear from our previous studies, for the first forty-eight or seventy-two hours. This indication is plainly fulfilled by free drainage. Fourthly, if possible, the secretions of wounds—which must be present to some extent—should be kept from chemical change, and rendered unfit for the generation of septic matters, by agents which, if possible, shall promote the formation of granulations. To sum up, the indications are: perfect coaptation, perfect drainage, disinfection, promotion of granulation, and rigid avoidance of any thing that can chemically or mechanically injure these safeguards of nature.

"Now, let us see how each one of the three typical methods of wound-dressing fulfils these indications, and whether the measure of success attained by each is not to be attributed to the degree of strictness with which they are carried out.

"Lister inculcates the most perfect coaptation, drainage, and disinfection which is practicable, with the least possible manipulation and after-dressing. Undoubtedly the carbolic acid, by coagulating the albumen in the tissues, forms a superficial, antiseptic, comparatively impermeable film, which, like that formed by chloride of zinc, remains until, by the formation of granulation, the slough—for such in truth it is—becomes separated. The antiseptic method certainly renders the wound-fluids unfit for the propagation of the septic poison; whether by preventing the formation of minute organisms or not, is beside our present purpose to consider.

"On the other hand, the 'open method' succeeds by very dissimilar methods in fulfilling some of the indications perfectly, while others it professedly and necessarily does not attempt. I say necessarily, because their fulfilment would inevitably prevent the carrying out of the others.

"Let us examine this method, as we have just done Listerism. The first indication, viz., as perfect coaptation as possible, is of necessity neglected, so that whatever advantage accrues from healing by primary union, is lost. In return for this, its advocates claim—and rightly—that the coaptation secured, although imperfect, is not disturbed by the accumulation of the discharges, and that they are prevented from remaining in contact with an absorbing surface for any considerable length of time, or in any quantity. By this removal, as soon as effused, of the wound-fluids, they maintain that chemical and septic changes are very unlikely, and should they obtain, do no harm, because the poison cannot remain in contact with an absorbing surface sufficiently long.

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[1] Experiments have proved that pressure favors absorption, so that this accumulation is dangerous on this account also.

"I admit that the second and third indications, which amount to free drainage to prevent separation of wound-surfaces, and free drainage to obviate the risk of absorption by the long retention of the wound-fluids in contact with absorbing surfaces, are admirably fulfilled, but the fourth indication is certainly not fulfilled, other than negatively, if the strictly open method is used. The disadvantages inherent to this plan will be presently adverted to. The advantages of this method are clear and unmistakable, but are not dependent upon any thing but the fulfilment of the principles pointed out, and upon which also depends the success of the antiseptic method.

"Finally, there remains for consideration the plan lately advocated by von Brun. Dispensing with the spray and the other elaborate formalities of the Lister operation, after its completion the stump is syringed out with large amounts, say half-a-gallon, of weak carbolized water; it is then dressed antiseptically, and left undisturbed for forty-eight hours. The dressings are then removed, and the stump treated by the open method. Subjected to the same critical analysis as the other two systems, we find that the most perfect coaptation possible, drainage by tubes, etc., prevention of chemical or other changes in the fluids, and treatment of the wound-surfaces by a coagulating agent, are relied upon, *and that this antiseptic condition is kept up for the first forty-eight hours*; in other words, until most of the primary danger is passed: because, as we have seen, healthy granulations are a protection against septic absorption, and their formation has commenced, and, in many cases, is well advanced by the time specified. At the end of the first forty-eight hours the troublesome details of the Lister antiseptic dressing are dispensed with, and the open method substituted. It is hardly necessary to point out that most of the good to be obtained by a Lister dressing is here combined with the advantages of the open method. In this plan, too, there is nothing mysterious, nothing occult, but the simple successful employment of certain well-established surgical principles.

"Look at the results of Alanson's famous series of major amputations performed nearly a century ago. He coaptated the deep surfaces of the flaps by compresses, leaving the edges comparatively to themselves. thus securing the inestimable advantages of quick healing, free drainage, and—because nothing could accumulate in amount—freedom from septic absorption. Examine the records of Callender's admirable series of amputations, and upon what did his success depend? Was it not upon free drainage, absolute quiet of the limb, treatment of the wound surfaces with a solution of chloride of zinc, thus forming an impenetrable antiseptic slough, which, upon separating some days after the operation, left a healthy granulating surface, and, finally, upon rigid avoidance of any thing that could injure the granulations, merely cleansing them with a camel's-hair pencil dipped—and kept in the intervals—in an antiseptic solution? The limb, where opportunities served, was swung for some days before the operation, which, of course, rendered the patient accustomed to the new position. This, in part, obviated those movements which Billroth has shown are apt mechanically to work the pus into the intermuscular spaces, where it can be absorbed, since it must then have broken down the barrier of the granulations.

"I maintain that the success of all these definite methods of wound-dressings,—indeed, the good result achieved by every surgeon, is necessarily dependent upon the degree in which he succeeds, wittingly or unwittingly, in fulfilling the indications pointed out in this paper.

"Provided Lister's directions are properly carried out, his method, though at first troublesome, seems to me both theoretically and practically the best. To operate satisfactorily it demands skilled assistance, some experience, and the utmost attention to detail, combined with a thorough appreciation of the principles involved. By excessive care upon the part of the operator, skilled assistance may be dispensed with, although it renders the labor greater, and risks the success of the operation. It is utterly unfair to deliberately transgress those rules upon which Mr. Lister lays most stress as being essential to success, and then condemn his method. He distinctly says that carbolic acid is an irritant to the tissues, and, therefore, by a special protective specially tested, he keeps it from direct contact with the wound; otherwise suppuration will occur. As the spray itself is an irritant—any operator's hands will demonstrate this,—special provision for draining off the excessive amount of serum poured out must be provided; yet, surgeons will almost hermetically seal a wound by stitches, straps, etc., thereby rendering suppuration from tension certain, when Mr. Lister has said that a single stitch producing tension will provoke suppuration. In this latter case, however, the pus does not become offensive.[1] Again, the less of the direct spray coming in contact with the raw surfaces the better, and yet we see a powerful atomizer within a couple of feet of an open abdominal cavity, the spray playing directly upon the exposed peritoneum, cooling it, if not doing other fatal damage, when the cry is again raised that Listerism is worse than useless. But what I have just described is *not* Listerism. The magnificent triumphs in recent abdominal surgery, considered by the most noted operators to be due to the use of this method, plainly demonstrate what the method in proper hands is capable of. An ovariotomy can be usually done with impunity, with strict Listerism, in a general hospital, where, without it, certain death has been proven to be the almost inevitable result."

In considering such an important subject as wound-treatment, no personal bias should be allowed to influence us. As Paget has remarked, an immense improvement in the success following operations had taken place before the advent of Listerism, and he thinks that experience has demonstrated that from 60–70 per cent. of patients are insusceptible to infective disease. That hospitalism is the only cause of pyæmia or septicæmia, and that mere cleanliness is the all-essential preventive, have been disproved by the statement of Prescott Hewitt and many other surgeons as to their experience in private practice, and I have myself witnessed it on more than one occasion. Admit all that the opponents of Listerism advance concerning cleanliness and hygiene, if Listerism will practically abolish infective diseases amid the worst surroundings, so that the results of operations at least equal those performed under the best circumstances, will not the application of the method with the most favorable surroundings prevent *all* that is preventable, viz.: all except those rare instances where blood poisoning has not its origin in the operation-wound?

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[1] During the hottest summer weather I have dressed a bad compound comminuted fracture of the leg, where profuse suppuration had resulted from inadequate drainage, at intervals of from *four* to *eight* days, without the faintest trace of putrefactive odor being perceptible. Other similar cases could be cited which have been treated by the author, but this will suffice to prove the truth of my statements.

Statistics can go but a short way to prove the advantages of any mode of treatment. The rapidity of recovery, condition of the wound, which is supple and non-indurated, the slight fever, the absence of necessity for change of diet, lessened drain, slight pain, etc., must be taken into consideration as favorable to asepticism, and if such results are considered desirable the method will sooner or later be adopted by the majority of surgeons.[1]

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[1] I have not attempted to refer to the various authors that I have mentioned specifically, but I here acknowledge my indebtedness to MacCormac, Van Buren, Cheyne, and others. The reader is referred for many additional facts and arguments elicited during the discussion following the reading of this paper. They will be found, in brief, in the *Phila. Medical Times* for July 29, 1882.

NOTES ON TWELVE CASES OF BRAIN TUMOR, CHIEFLY WITH REFERENCE TO DIAGNOSIS.*

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THE notes presented are on twelve cases of intracranial tumor, in which the clinical and *post-mortem* examinations were made by myself. Five of the cases have already been published in detail in various journals, to which references are made below; seven have not before seen the light of publication. I have endeavored to condense the clinical histories and the *post-mortem* and microscopical notes into the narrowest limits compatible with an intelligent understanding of the cases.

CASE I.—*Clinical History:* Male, æt. 35. History of syphilis. Fell from door-step, striking head. Headache persistent and violent. Vertigo. Mental slowness and uncertainty; attention destroyed; hysterical sobbing and crying. Nystagmus; tonic spasm in muscles of neck and forearm. No true paralysis, but movements weak and uncertain. Speech explosive. Blindness which came on gradually, first in right eye then in left. Conjunctivitis in left eye; corneitis with tendency to sloughing; sensibility of conjunctiva and cornea lowered. Choked disks. Smell defective. Polyphagia. Constipation, alternating with involuntary evacuations. Urine passed involuntarily. Temperature from 97° to 101° F.; higher in the evening. Head-temperatures above normal; highest at frontal station, averaging 96.5° F.

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* Read before the American Neurological Association, June, 1882.

Pathological Anatomy: Fibroma. In the right frontal lobe, involving the anterior upper half of the first frontal convolution, the anterior upper and inner half of the second frontal, a small segment of the gyrus fornicatus just where it bends round the knee of the corpus callosum, and the anterior half-inch of the corpus callosum itself. The membranes over the tumor were destroyed. Microscopical sections from the posterior part of the eyes at the embrace of the optic nerve presented the appearance usually encountered in choked disks.

Remarks: Philadelphia Medical Times, January 18, 1879, in Proceedings of the Pathological Society of Philadelphia.

CASE 2.—*Clinical History:* Male, æt. 36. History of syphilis. Headache, continuous and nearly always excruciating. Head sensitive to pressure everywhere, but most in frontal and left parietal regions. Faculty of attention wanting; continuous mental action impossible; often crying. General hyperæsthesia. Sight impaired; pupils contracted. No opthalmoscopic examination. Constipation. Fever with delirium; finally sopor and coma. During last week of life the pulse ranged from 90 to 110; temperature from 100° to 101.5° F.; respiration from 20 to 34.

Pathological Anatomy: Three gummata. (1) The largest growth, $1\frac{1}{4}$ in. in diameter, adherent to pia mater, in the right præfrontal region, involving the superior anterior portion of first and second frontal convolutions. Pia mater adherent to dura over tumor; a cheesy mass between dura and bone; strong adhesions between dura mater and skull; some surrounding pachymeningitis. (2) A growth, $\frac{3}{8}$ths of an inch in diameter, adherent to pia mater, which was fused with the dura, in the retrocentral fissure, $\frac{1}{2}$ inch behind the junction of the upper and middle third of the fissure of Rolando. (3) A growth $\frac{5}{8}$ths of an inch in diameter, adherent to pia mater, in the supramarginal convolution, at the extreme posterior point of the horizontal branch of the Sylvian fissure. Dura mater adherent to the calvarium at several points over both hemispheres.

Remarks: Not before published.

CASE 3.—*Clinical History:* Female, æt. 35, white. History of syphilis. Headache, severe, at first with long intermissions; later continuous, and often agonizing. Vomiting with paroxysmal headache, coming on late in illness. Vertigo, usually with headache. Excitable and irritable. Slight twitching of mouth, hands, and forearms. Left hemiplegia; upper as well as lower fibres of facial nerve paralyzed. Left internal strabismus. Electro-contractility diminished. Difficulty in enunciation. Sensibility diminished in left leg. Late hyperæsthesia, and great pain in paralyzed limbs. Conjunctivitis and necrosis of cornea of left eye; conjunctiva and cornea insensitive. Sight impaired. Descending optic neuritis. Hearing impaired on left side. Smell defective. Profuse perspiration, more marked on right side than on left. Constipation. Temperature, taken in right and left axilla for eleven weeks before death, gave the following averages: Right axilla, 99.1°, M.; 100°, E. Left axilla, 99.4°, M.; 101.4°, E. On some days remarkable falls in temperature to 96°, 95°, and even 94° and 93°. Average head-temperatures above the normal: for stations on right side of head averaging about 97°; on left side, about 94.3° F.

Pathological Anatomy: Gumma. A twin-tumor, each mass about ⅔ds of an inch in diameter, in front of the optic chiasm. The growths involved the basal termination of the corpus callosum, the peduncles of the corpus callosum, the lamina cinerea, and anterior perforated spaces. They also probably encroached upon the roots of the olfactory nerves, the optic nerves and commissure, and the anterior portions of the circle of Willis, which seemed to have been broken in front. The base of the brain, from the posterior line of the tumor backward to the pons, was markedly softened. Microscopical sections of optic nerves showed the appearances peculiar to descending neuritis in a somewhat advanced stage.

Remarks: Philadelphia Medical Times, March 23, 1879, in Proceedings of the Pathological Society of Philadelphia. Also *New York Medical Record*, August 9, 1879.

CASE 4.—*Clinical History*: Female, æt. 30. No history of causation. Headache, continuous, sometimes agonizing. Percussion of head caused most pain in right parietal region. Vomiting when headache was most severe. Vertigo. Mind clear, but acted slowly; emotional. Spasm, beginning with twitching of fingers of left hand; most severe on left side, and especially in left arm. Upper as well as lower fibres of left facial nerve partially paralyzed; nearly complete paralysis of left arm; slight paralysis of left leg. Bowels and bladder partially paralyzed. Impaired sensibility in limbs of left side. Left patellar reflex diminished. Sight very imperfect. Choked disks. Hearing defective in right ear.

Pathological Anatomy: Carcinoma. About 1½ inches in diameter beneath and adherent to the pia mater of the convexity of the right hemisphere; the pia and dura mater were united by strong adhesions. The tumor involved the middle portion of the ascending parietal convolution and the upper part of the inferior parietal lobule, pushing aside the interparietal fissure. The anterior extremity of the tumor was about ⅕th inch back of the centre of the fissure of Rolando. On the inner side of the tumor the white matter of the brain was broken down.

Remarks: Reported at the meeting of the American Neurological Association, June, r881, and published in ARCHIVES OF MEDICINE, August, 1881.

CASE 5.—*Clinical History:* Female, æt. 38. History of syphilis. Blows on the head. Headache, with agonizing paroxysms. Top and right side of head sensitive to percussion; and headache severest in these regions. Vomiting. Vertigo. Great mental irritability. Severe left-sided spasm, beginning with twitchings in left toes and foot. Partial paralysis of right leg and arm, most marked in leg. Hyperæsthesia. Impaired sight. Choked disks. Head temperature taken once: right parietal region, 97.2° F.; left parietal region, 96° F.

Pathological Anatomy: Gumma. Attached to the fused membranes of the right convexity. Involved the upper fourth of the ascending frontal, and a smaller segment of the ascending parietal convolution, crossing the fissure of Rolando at its upper extremity. A good example of strictly cortical lesion, involving only gray matter, and having only a very thin layer of softened tissue on its inner side. Microscopical examination of the optic nerves showed the appearances of choked disks, with probable ascending neuritis.

Remarks: Not before published.

CASE 6.—*Clinical History:* Male, æt. 31. Evidences of tuberculosis. Headache, continuous, with severe exacerbations; most severe at vertex. Vertigo. Some irritability and emotionality; hallucination that some one was going to come and take him away. Spasm confined to left arm. Partial paralysis of left arm and leg, and late in his illness, of left side of face. Left hemianæsthesia, at first partial, but later complete and persistent. Sight impaired; right pupil dilated and left contracted before death. No ophthalmoscopic examination. Hearing defective in left ear; tinnitus aurium. Head-temperature taken once: right frontal region 98° F.; left frontal region, 96.3° F. Cheyne-Stokes breathing on day of death.

Pathological Anatomy: Tubercular tumor. Dura and pia mater adherent over the tumor, which involved the posterior extremities of first and second frontal, and upper thirds of both ascending convolutions, of right hemisphere. Interior of hemisphere broken down into a cavity containing brain detritus and some pus ; the parts destroyed included white matter of the parietal lobe, the posterior third of the lenticular nucleus, and the adjacent posterior portion of the internal capsule. Miliary tubercles in pia mater around and near the tumor.

Remarks : Not before published.

CASE 7.—*Clinical History :* Male, æt. 25. Kicked on the head. Headache, nearly continuous, with violent paroxysms. Vomiting at intervals, most when headache was most violent. Vertigo. Mental confusion ; sometimes maniacal. Left convergent strabismus. Partial right hemianæsthesia. Patellar reflexes slightly exaggerated ; slight ankle clonus on the right side. Blind in both eyes ; sight of right eye lost first. Choked disks, and eventually optic atrophy. Hearing defective in right ear. Constipation. Head-temperatures taken once : right parietal region, 98° F.; left parietal, 97.8° F.

Pathological Anatomy : Fibroma. A firm growth, 2½ inches in diameter, adherent to membranes, and involving the left occipital and postero-parietal region to within a half inch of longitudinal fissure. The brain tissue around and beneath the growth was softened and broken down, the parts disintegrated being chiefly the white matter of the postero-parietal and occcipital lobes.

Remarks : Not before published.

CASE 8.—*Clinical History :* Male, æt. 33. History of syphilis. Blows on the head. Headache, continuous, usually dull, occasionally severe. Vomiting occasional. Stupidity, want of energy, drowsiness. One general convulsion a few hours before death. Partial anæsthesia on the right side. Hyperæsthesia of left side. Sight impaired. No ophthalmoscopic examination. Hearing defective in right ear. Constipation.

Pathological Anatomy : Gumma. Dura and pia mater adherent. Tumor 1¼ inches in diameter, attached to adherent membranes, and involved the middle portions of the first and second temporal convolutions of left hemisphere. A layen of brain substance both anterior and posterior to the tumor was softened. The tumor grazed, but did not involve, the ascending parietal and inferior parietal convolutions. A large *foyer* of greenish-yellow pus was found to the inner side of the tumor, the abscess having destroyed a considerabe portion of the white matter of the temporal lobe.

Remarks: Not before published.

Case 9.—*Clinical History:* Female, æt. 13. Tubercular history. Headache, with severe paroxysms; most marked in frontal and occipital regions. Vomiting. Vertigo; attacks of reeling and falling. Mental dulness. Weakness of limbs, but no distinct paralysis. Hyperæsthesia; severe pains in limbs. Gradually went blind, first in left eye, and then in right. Descending neuritis determined nearly a year before death; marked optic atrophy observed a month before death. Constipation.

Pathological Anatomy: Tubercular tumor. A large nodulated mass which occupied the lower two thirds of the right cerebellar hemispheres; one small nodule extended across the posterior extremity of the superior vermiform process, destroying altogether about one third to one half of a cubic inch of its substance. Internal hydrocephalus, the ventricles and the horns of the lateral ventricles being greatly dilated and filled with fluid.

Remarks: Not before published.

Case 10.—*Clinical History:* Female, æt. 27. History of syphilis. Headache, with paroxysmal exacerbations. Vomiting, frequently recurring; for four weeks before death vomited almost incessantly. Vertigo; impossible to sit or stand because of vertigo late in illness. Slowness of mind, and hebetude. General muscular weakness. Right partial hemiplegia and hemianæsthesia. Trigeminal neuralgia. No ophthalmoscopic examination; but sight impaired. Marked slowness of respiration; respirations ran down as low as four and five per minute two weeks before death.

Pathological Anatomy: Nature of growth not determined. A nodulated mass, ¾ths of an inch in greatest diameter, involving the right middle cerebellar peduncle and the adjacent region of the right cerebellar hemisphere. The right upper half of the floor of the fourth ventricle was superficially softened. An area of softening was also found involving a small portion of the under outer surface of the left optic thalamus, the entire breadth of the internal capsule, and a small segment of the lenticular nucleus.

Remarks: Not before published.

Case 11.—*Clinical History:* Male, æt. 32. Thrown from a horse and kicked on the head. History of syphilis; headache, severe at first, always came on at night; became less severe later. Vertigo. Defective memory; apathy. Right hemiparesis; helplessness of all the limbs before death; partial ptosis of the right side. Sensation diminished in the left side of the face and in the right limbs. Pupils small before death. Descending optic neuritis. Conjugate deviations of the eyes and rotation of the head to the right. Persistent epistaxis ; tendency to hemorrhage from mucous membranes. Constipation.

Pathological Anatomy : Gumma and fibroma. (1.) Gumma, half an inch in diameter, distinctly limited to the left upper quarter of the pons. (2.) Fibroma, no larger than a pea, between the dura and pia mater,causing a slight depression in the first temporal convolution about the junction of its middle and posterior thirds, and half way between the parallel fissure and the horizontal branch of the Sylvian fissure. Microscopical examination of the optic nerves showed a descending neuritis of subacute character.

Remarks : Reported at the meeting of the American Neurological Association, June, 1881, and published in the *Journal of Nervous and Mental Disease*, July, 1881.

Case 12.—*Clinical History :* Male, æt. 35. History of syphilis. Wounded in head by glancing bullet. Headache of great severity at intervals. Vomiting at time of headache. Vertigo. Failure of memory and depressed spirits. Epileptiform attacks ; in the spasms lifted up the right side of his body and worked over toward the left. Occasional cramps in stomach and legs. Temporal and orbital neuralgias. Descending neuritis, and eventual atrophy of both optic nerves. Constipation. Died at the close of a series of convulsions, death being preceded by general paralysis.

Pathological Anatomy : Gumma. A rounded mass, quarter of an inch in diameter, attached to the pia mater, just to the left of the centre of the anterior surface of the pons. The entire anterior central portion of the pons was softer and more doughy than usual. At the junction of the pons with the medulla oblongata was a recent hemorrhage, which had spread downward to about the middle of the latter.

Remarks : Brain, January, 1880.

I will recapitulate and summarize, with a few remarks, some of the most important facts brought out in the foregoing reports :

Etiology.

History of syphilis in three cases.
" " " and traumatism in five cases.
" " traumatism alone in one case.
Tubercular history in two cases.
No history in one case.

Pathological Anatomy.

Gummata in six cases.
Fibromata in two cases.
Tubercle in two cases.
Carcinoma in one case.
Not determined in one case.

The General Diagnosis of Tumors of the Brain.—I believe that the general diagnosis of the existence of intracranial tumors can be made with greater certainty than that of almost any other serious encephalic disease. It is true that cases have been reported in which autopsies have revealed the presence of large tumors, which are said to have caused no symptoms during life; but I cannot get rid of the impression that such cases have been more or less imperfectly observed and recorded. The symptoms which point conclusively to the existence of an intra-cranial tumor are as follows:

1. Headache.
2. Pain on percussion of the head.
3. Vomiting.
4. Vertigo.
5. Mental disturbances.
6. Hyperæsthesia.
7. Choked disks or optic neuritis.
8. Elevated temperature of the head.
9. Constipation.

Headache.—Headache was present in every case. In ten cases more or less headache was present all the time; and in all of these paroxysms of agonizing severity occurred. Absolute intermissions in the headache were observed in only two cases, and in one of these the pain became continuous before the end. The usual type of headache from intracranial tumor might be described as a continuous pain, sometimes of persistent severity, and generally with exacerbations of great violence. In two instances the patient complained spontaneously of the pain being greatest in the region of the head nearest to the seat of the growth. In one of the cases of cerebellar tumor the patient referred the pain sometimes to the occipital and sometimes to the frontal region. Mr. Callender has noticed that cortical lesions, like those observed in these cases, are more frequently accompanied by localized pain than lesions of deeper parts (*Bartholomew's Hospital Reports*, 1869, and Ferrier's "Localization of Cerebral Disease," p. 99).

Percussion of the Head.—In three cases percussion of the head elicited an intensified pain in the region corresponding to the location of the tumors. As this procedure was only resorted to in four cases, the results favor the view that percussion of the skull may afford valuable aid in localizing encephalic tumors, particularly when they are meningeal or cortical. Ferrier (*Brain*, January, 1879) believes that percussion often elicits pain over the cranial region beneath which a lesion is localized. Dr. Alex. Robertson, of Glasgow (*Abstr. Internat. Med. Congress*, 1881, p. 85, and *Journal of Nervous and Mental Disease*, April, 1882), has also directed attention to the value of percussion of the skull in the localization of disease on the surface of the brain.

Vomiting.—Vomiting was a symptom in eight of the twelve cases. In Cases 1 and 2, in which the chief lesions were in the antero-frontal regions, vomiting was not present, although the tumors were of considerable size, and the other manifestations were of the most decided character. Even in Case 3, in which the tumor was well forward but low down, vomiting did not come on until late. In Case 6, a tubercular growth of the postero-frontal region, vomiting was absent; although in Cases 4 and 5, in which the lesions were in the motor zone near by, it was a prominent symptom. In both cases of cerebellar tumor, vomiting was prominent; one of these (Case 10) was the most remarkable example of persistent cerebral vomiting which it has ever been my lot to observe. For four weeks before her death she vomited almost continuously.

I incline to agree with Ferrier (*Brain*, July, 1879) that the majority of cases of cerebral vomiting can be ascribed to irradiation of irritation of the nerves of the cerebral membranes, or to the physical effects of acute pain. The nuclei of the trigeminus and of the vagus are in close anatomical relationship in the medulla oblongata. Irritative impressions conveyed from the cerebral membranes, and particularly from the dura mater, to the trigeminal nuclei in the medulla, are transferred to the vagus and give rise to vomiting. In tumors of the cerebellum the vomiting may be brought about through the affection of the centres of equilibration, and the concurrent vertiginous sensations (Ferrier); or it may be due to direct irritation by contiguity of the floor of the fourth ventricle. In general, the tumors situated farthest forward in the brain appeared to be the least likely to give rise to vomiting.

Vertigo.—Vertigo was noted in ten cases. Under this general designation dizziness, giddiness, reeling, insecurity in the erect posture, etc., have been included. The vertigo which so constantly accompanies brain tumors can be explained in several ways besides the view of the affection of centres of equilibration in the cerebellum. Like headache and vomiting it seems sometimes to flow from pure irritation of the cerebral membranes. Alterations in the intercranial circulation produced by the growth of the tumor, is another probable cause. The experiments of E. Weber have demonstrated the existence of a communication between the arachnoid cavity and the labyrinth, and, consequently, the increased intra-cranial tension present may result in pressure on the labyrinthine fibres of the auditory nerve, and thus occasion vertigo.

Mental Disturbances.—Mental disturbance of some sort occurred in almost every case. When I say this, however, I am using the term mental disturbance in a very general sense. The majority of the patients were emotional, excitable, and irritable. It was not uncommon for them to sob and cry apparently from the great pain. They were usually easily affected by external impressions. One case (6) showed marked hallucinations of fear. Another case (7) had at times maniacal attacks, which usually coincided with the most violent paroxysms of headache. Some of the cases exhibited dulness, stupidity, want of energy, or apathy. Failure of memory, depression of spirits, and hebetude were other mental manifestations. Mental slowness and uncertainty, inability to fix the attention, and impossibility of continuous mental action were strikingly shown, particularly in the cases of tumor in the præfrontal regions.

Hyperæsthesia.—Hyperæsthesia was noted in five cases. It is a common symptom in brain tumor; sometimes it is general, more frequently it is present in the limb or limbs affected with spasm or paralysis. With the hyperæsthesia, the patients not infrequently complain of severe spontaneous pains in the limbs.

Choked Disks and Optic Neuritis.—Ophthalmoscopic examinations were made in eight cases. In four of the eight, microscopical examinations of the eyes and the optic nerves were also made. I wish to acknowledge special obligations to Dr. E. O. Shakespeare, pathologist and ophthalmologist to the Philadelphia Hospital, by whom the ophthalmoscopic and microscopic examinations were conducted in these four cases. The results in the eight cases were as follows:

Choked disks in four cases.

Descending neuritis in four cases.

Both eyes were affected in each case. In every case, therefore, in which the eyes were examined with the ophthalmoscope, marked changes were found. It will be seen that, with Allbutt and others, I make a distinction between choked disks, or ischæmia of the disks, and true descending neuritis. A brain tumor may give rise to either of these conditions, or to both in the same patient.

A careful examination of the reports will show that true choked disks were present in tumors of the convexity. In three of the cases of descending neuritis the tumors were situated at the base, and the fourth was a tumor of the cerebellum with hydrocephalus. Ischæmia of the disks may also have been present at some stages of these cases. Descending neuritis would seem to be most likely to occur when the tumors are so situated that the inflammation set up by them can readily extend by anatomical continuity to the optic nerves. The "lymph-space theory" seems to me to best account for the occurrence of choked disks in cases of tumor situated remotely from the base. "According to this theory, owing to increased intracranial pressure, partial stasis occurs in the veins, with exudation of the watery parts of the blood into the subdural and subarachnoid cavities, and this fluid, seeking the point of least resistance, finds its way into the lymph space between the outer and inner sheaths of the optic nerves, and, by gradual pressure, causes a dilatation of the peripheral end of the dural sheath just before it passes into the sclerotic, compressing, at the same time, the pial sheath and the nerve fibres covered by it."[1] The presence of choked disks or optic neuritis should be regarded as the strongest possible corroborative evidence of the existence of an encephalic tumor.

Temperature of the Head.—Observations on the temperature of the head were made in five cases (Cases 1, 3, 5, 6, and 7). Full accounts of the observations in two of these cases (1 and 3) have already been published (*Philadelphia Medical Times*, January 18, 1879, and *New York Medical Record*, August 9, 1879). It is not within the scope of the present paper to review the general subject of cranial or cerebral thermometry. I will simply, in passing, recall to the Association the labors of E. Seguin,[1] Broca,[2] Gray,[3] Lombard,[4] Maragliano and Seppilli,[5] and Amidon.[6]

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[1] Optic Neuritis as a Symptom of Intracranial Disease, by William F. Norris, M.D., *Philadelphia Medical Times*, August 30, 1879.

Dr. L. C. Gray (*N. Y. Medical Journal*, August, 1878) has recorded some observations in cerebral thermometry in one case of tumor of the brain. The tumor, a soft, jelly-like mass the size of a hazel-nut, was found between the horizontal branch of the Sylvian fissure and the first temporal fissure, while the whole of the right occipital lobe was converted into a colloid, extremely vascular mass, which gave way under examination, this degeneration also extending anteriorly to the tumor as far as the fissure of Sylvius. Microscopical examination showed the tumor to be a typical glioma.

The temperatures at different stations were as follows:

	Left.	Right.
Frontal	96.75°	98.33°
Parietal	95°	99.75°
Occipital	96.75°	100.50°

The average of the left side was 96.16°; of the right, 99.52°; of the whole head, 97.84°.

In my case of frontal tumor, reported in the *Medical Times* (Case 1), the average temperatures of the different stations taken were as follows:

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[1] "Medical Thermometry and Human Temperature," by E. Seguin, M.D., William Wood & Co., New York, 1876.

[2] *Progrès Médical*, 1877.

[3] *New York Medical Journal*, August, 1878.

[4] "Experimental Researches on the Temperature of the Head," London, H. K. Lewis, 1881.

[5] Quoted in ARCHIVES OF MEDICINE, 1879.

[6] ARCHIVES OF MEDICINE, April, 1880.

Middle frontal station	96.5°
“ occipital “	95·5°
Right frontal “	95°
Left “ “	94·7°
Right parietal “	94·7°
Left “ “	94·4°

In the case reported in the *New York Medical Record*, August 9, 1879 (Case 3), a gumma which involved the basal termination of the corpus callosum, the peduncles of the corpus callosum, the lamina cineria, and anterior perforated spaces, the average temperatures at the different stations were as follows:

Right frontal station	97·1°
Left “ “	97·4°
Right parietal “	97.6°
Left “ “	94°
Right occipital “	97°
Left “ “	94·5°
Right anterior vertical station	97°
Left “ “ “	94°
Right posterior “ “	96.6°
Left “ “ “	94·3°

The normal average temperatures, as determined by Gray, were as follows:

Left frontal station	94·36°
Right “ “	93·71°
Left parietal “	94·44°
Right “ “	93·59°
Left occipital “	92.66°
Right “ “	91.94°
Left side of the head	93·83°
Right “ “	92.92°
The whole head, exclusive of the vertex	93·51°
Motor region of the vertex	91.67°
The whole head, inclusive of the vertex	92.66°

In Case 5, a gumma in the cortical motor zone of the right side, the head-temperatures, taken once, were found to be for the right parietal region 97.2°; for the left, 96°. In Case 6, also a tumor of the right motor zone, the right frontal region gave a temperature of 98°; the left, of 96°. In Case 7, a tumor of the left occipito-parietal region, the temperatures were for the right parietal region, 98°; for the left, 97.8°. Although the temperature of the head was only taken once in each of these three cases the observations were carefully made, with tested thermometers.

I conclude, from a study of the results obtained in these cases, that in brain tumors the average temperature of the whole head is elevated several degrees above the normal; and that the elevation of temperature is usually greatest at the station nearest the seat of the growth.

Constipation.—Constipation was recorded in eight cases. In some instances the bowels, after having been obstinately closed for days, would open involuntarily. In the terminal periods, involuntary evacuations both of the bowels and bladder often occur, but a real diarrhœa is rare.

Miscellaneous Points.—Severe *neuralgic* pains in the limbs were complained of by one of the cases of cerebellar tumor (Case 9). The other case of tumor of the cerebellum suffered from trigeminal neuralgia. One case of tumor of the pons Varolii had frequent and severe attacks of temporal and orbital neuralgia. Extreme trigeminal neuralgia, in association with the other symptoms of brain tumor, would seem to point to the pons or cerebellum as the probable seat of the growth. A study of the *reflexes* was unfortunately neglected in most of the cases. In Case 4, however, a carcinoma of the right motor zone, the left patellar reflex was diminished; while in Case 7, an occipito-parietal tumor of the left hemisphere, the patellar reflexes were a little exaggerated, and even slight ankle clonus could be brought out on the right side. The persistent *epistaxis* and tendency to *hemorrhage from the mucous membranes* were interesting vaso-motor phenomena in the case of tumor situated in the left upper quarter of the pons (Case 11). Profuse *perspiration*, more marked on one side, was noted in Case 3. *Polyphagia* was present in one (Case 1). *Disturbances of respiration* were observed in several cases. In Case 6, a tubercular tumor of the right motor zone, with breaking down of the interior of the hemisphere, Cheyne-Stokes breathing occurred on the day of death. Extraordinary slowing of respiration occurred in Case 10, a tumor of the right middle cerebellar peduncle and cerebellar hemisphere, which caused irritation and softening of the floor of the fourth ventricle. *Hysterical manifestations* were noted in several instances, and are of not infrequent occurrence. Cases 1 and 3 exhibited *inflammatory, trophic, and anæsthetic phenomena* in the eyes. In both cases the growths were in the antero-frontal region, one extending to the base, and the other not involving the orbital surface. It is worthy of remark with reference to Case 1, that the tumor was in the *right* præfrontal lobe, and the *left* eye was attacked. *Smell* was observed to be defective in Cases 1 and 3. *Defective hearing* was noted in five cases. In Case 3, a tumor in front of the optic chiasm, hearing was defective in the left ear, the same side as the paralysis and other marked symptoms. Softening at the base was present in addition to the tumor. In Case 4 it was defective in the right ear, the tumor in this instance being situated in the right motor zone. The white matter of the brain on the inner side of the tumor was broken down. In Case 6 hearing was impaired in the left ear. The tumor in this case involved the posterior extremities of the first and second frontal and upper thirds of both ascending convolutions of the right side; and white matter of the parietal lobe, the posterior third of the lenticular nucleus, and the adjacent posterior portion of the internal capsule were softened. In Case 7, a tumor of the left occipito-parietal region, hearing was defective in the right ear; and in Case 8, a tumor and abscess of the left temporo-sphenoidal lobe, it was also impaired in the right ear. These last three cases would seem to give some support to the localization of auditory centres in the temporo-parietal region.

The Local Diagnosis of Tumors of the Brain.—Niemeyer would hardly say to-day that "the 'brilliant diagnoses', where the precise localization of a tumor is fully confirmed by autopsy, are not usually due to acumen of the observer, but are cases of lucky diagnosis."

The locations of the tumors in the twelve cases may be summarized as follows:

Antero-parietal region . . .	3 cases.
Rolandic " . . .	3 "
Parieto-occipital " . . .	1 case.
Temporo-sphenoidal lobe . .	1 "
Cerebellum	2 cases.
Pons Varolii	2 "

Tumors of the middle portion of the base of the brain, the region of origin of the various cranial nerves, can, of course, be diagnosticated with comparative ease by a study of the various forms of paralysis and spasm in the distribution of these nerves, in connection with other special and general symptoms. In several cases this region was more or less involved. Varieties of alternate hemiplegia are to be looked for, and also isolated or associated palsies of the oculo-motor, pathetic, facial, trigeminal, and other cranial nerves. In studying these palsies, it must be borne in mind that although the lesions producing them are intracranial, the paralyses themselves are peripheral.

Tumors of the Antero-Frontal Region.—Tumors of the præfrontal lobes can be pretty certainly diagnosticated, partly by a study of the actual symptoms observed, and partly by a process of exclusion. Headache of the usual type, vertigo, choked discs, inflammatory and trophic affections of the eyes, widely varying body-temperature, and high head-temperature are among the most positive manifestations. Mental slowness and uncertainty seem to be greater in these cases than in others. The absence of true paralysis and of anæsthesia is characteristic. Nystagmus and spasm in the muscles of the neck and forearm were present in one instance, but usually marked spasm is not to be expected. Vomiting would seem to be less frequent than in cases of tumor situated farther back.

Tumors of the Rolandic Region, or Motor Zone of the Cortex.—Tumors of the motor zone of the cerebral cortex, the region surrounding and extending for some distance on each side of the fissure of Rolando, can be diagnosticated with great positiveness. Three of the twelve cases were examples of tumors of this region, and in each case the diagnosis of the location of the growth was accurately made during life. Seguin[1] gives the following symptoms as characteristic of tumors in the motor zone of the hemispheres: Localized convulsions in peripheral muscles; equally localized paralysis of peripheral parts; neuro-retinitis or choked disk; localized headache. To these I would add pain elicited or increased by percussion of the head near the seat of the tumor, and elevated temperature of the head, particularly in the region corresponding to the position of the growth.

The spasmodic symptoms usually precede the paralysis in these cases. The spasm is often local and generally begins in the same part in different attacks,—in the fingers, or toes, or face of one side.

Tumors of the Parieto-Occipital Region.—Tumors of the parieto-occipital regions present some characteristic peculiarities. Case 8 was the only one in which the tumor was located in this region; but in several others the white matter of the parietal lobe was softened as the result of the obliteration of blood-vessels by the tumors. In general terms, I might say that hemi-anæsthesia, partial or complete, and impairment of sight and hearing, on the side opposite to the lesions, seemed to be the most constant peculiarities.

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[1] On the Early Diagnosis of some Organic Diseases of the Nervous System, by E. C. Seguin, M.D., *N. Y. Medical Record*, Feb. 26, 1881.

Tumor of the Temporo-Sphenoidal Lobe.—The single instance of tumor and abscess of the temporo-sphenoidal lobe (Case 8) was not very closely studied. Besides the headache, vomiting, stupidity, and constipation, the only symptoms which seemed characteristic were the partial anæsthesia and defective hearing on the side opposite to the lesion. Sight was impaired, but a comparative study of the condition of vision in the two eyes was not made.

Tumors of the Cerebellum.—The diagnosis of tumor of the cerebellum can be made with considerable certainty, although, as a rule, not much will be learned of the functions of the cerebellum through a study of such cases. The symptoms are, in large part, the result of pressure and irritation of adjacent regions—of the floor of the fourth ventricle, of some of the cranial nerves, etc.

In the two cases of tumor of the cerebellum given in the list, headache, vomiting, vertigo, mental dulness or slowness, general muscular weakness, and impaired sight, were present. Right partial hemiplegia and hemi-anæsthesia were observed in one case, but these symptoms were doubtless due to an area of softening found in the left optic thalamus, internal capsule, and lenticular nucleus. Hyperæsthesia and severe pains in the limbs were marked in one of the cases. In one vomiting was almost incessant for four weeks. Vertigo in this case was also intense; late in her illness the patient could not raise herself from a recumbent position owing to the vomiting and vertigo. The cases were seen at too late a period to study carefully the peculiarities of the vertigo, and special disorders of movement. The slow rate of respiration in one case was quite remarkable.

In one of the cases internal hydrocephalus was present. According to S. Mackenzie, tumors of the cerebellum are very liable to be complicated by effusion into the ventricles of the brain, due either to pressure on the venæ magnæ Galeni, or to obliteration of the communication between the spinal and cerebral subarachnoidal spaces. (Ross on "Diseases of the Nervous System," volume ii, page 673.)

I agree with Nothnagel that a study of cases of atrophy of the cerebellum would be much more likely to throw light upon the functions of the brain than an investigation of cases of tumor and other lesions, which may cause functional disturbances of neighboring regions.

Tumor of Pons Varolii.—A large tumor of the pons Varolii can always be positively localized. Smaller growths may be localized with considerable definiteness in each of the four quarters of the pons. Alternate hemiplegia of the facial type was not present in either of the two cases of tumor of the pons, a fact which is explained by the comparatively small size and peculiar locations of the growths. In Case 11, the tumor, one half inch in diameter, was distinctly limited to the left upper quarter of the pons. The chief symptoms diagnostic of location were right hemiparesis and ptosis; partial anæsthesia of right limbs and left side of face; and conjugate deviation of the eyes and rotation of the head to the right. The other usual symptoms of tumor, with the exception of vomiting, were also present. It would seem that tumors limited to one half of the upper portion of the body of the pons will cause conjugate deviation of the eyes and rotation of the head away from the side of the lesion. This symptom may be due to an involvement of the fibres of the middle cerebellar peduncles which pass through the pons. In Case 12, in addition to the headache, vomiting, vertigo, mental failure, constipation, and descending neuritis, the diagnostic signs were trigeminal neuralgia, and spasms most marked on the right side of the body.

(*To be continued.*)

EDITORIAL DEPARTMENT.

THE NEW DEPARTURE AT THE ROOSEVELT HOSPITAL.

At last one of the important general hospitals of New York has organized a continuous surgical service. This unquestionable advance has been brought about, at this late day, by a variety of causes, and we believe that it will mark an epoch in the history of medicine in this city.

For four or five years the time-honored system of rapidly rotating medical and surgical services, each physician or surgeon being on duty for two or three months, has been severely criticized in various ways.[1] This absurd system, which operated to the detriment of patients, which tended to demoralize the young *internes* of the hospitals, and which seriously crippled hospitals as centres for education, was perpetuated, we believe, mainly to allow a large number of physicians to enjoy the honorable title of physician or surgeon to a hospital, and to economize their time for private practice ; and it is not surprising that the present beginning of reform should have been brought about by the persistent efforts of a small minority of the large number of the hospital physicians of New York. "To the victors belong the spoils," is sometimes a good saying, and we are glad that the new continuous service at the Roosevelt Hospital has come into the hands of one who by his pen and by his personal appeals has probably done more than any one else to bring about the change.

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[1] Consult Report of the Medical Board of the New York Hospital as to the Internal Administration of the Hospital, pamphlet, 1876. Editorials in ARCHIVES OF MEDICINE, vol. v, p. 177, and p. 291 ; vol. vi, p. 57.

One of the first practical attempts to improve the medical and surgical services of our hospitals was the simultaneous action of the governors of the New York Hospital and the trustees of the Roosevelt Hospital in 1878. This action consisted in the adoption of resolutions which provided that whenever by natural causes, such as death or resignation, vacancies occurred in the medical and surgical staffs of these institutions, these vacancies should not be filled until the number of each staff was reduced to three physicians and three surgeons: and, further, that in future, no newly appointed physician or surgeon could serve in both hospitals. Thus a blow was struck at the evil so prevalent in New York, of medical men holding appointments in two or three hospitals.

It is pleasant to add that a small majority of the medical staff of these two hospitals were heartily in favor of this movement, and urged their trustees and governors to take the decisive step.

At the Roosevelt Hospital, by the operation of these resolutions, the staff of attending surgeons was very soon reduced to two members, and the beginning of this year (1882) saw the entire surgical service of this important hospital under Dr. H. B. Sands and Dr. Erskine Mason. This division embraced a total of seventy-two beds, with an average occupancy of fifty patients. One would naturally suppose that a very satisfactory solution of the problem had been reached by the assignment of the two surgeons named, either to have the whole service for alternate periods of six months, or, better, by making two continuous services of thirty-six beds each. This latter plan would have been preferable, if complemented by the creation of a second staff of surgical *internes*, giving each surgeon a full corps of assistants. The extra expense entailed upon the hospital by the board and lodging of these gentlemen, would, in my opinion, have been compensated by the increased attention received by patients, and by the education of thrice the number of resident pupils to serve the community afterward. It is useless to speculate upon the manner in which this service might have been properly divided, since the problem, which apparently presented some difficulties, was abruptly solved by the death of Dr. Mason, in April. In him the hospital lost a faithful and distinguished surgeon, and the profession one of its worthiest members.

By this event, Dr. Sands was left the only attending surgeon of the hospital, and the trustees were called on to decide upon the appointment of a successor to Dr. Mason. The medical board of the hospital, by a majority of one, resolved to recommend to the trustees the names of certain candidates for the vacant position. Three members of the medical board thought that the service should have two surgeons, and two members thought that it would prosper better under one head. This difference of opinion was a sincere one, and each side could bring good arguments in support of its view of the situation.

The trustees of the hospital were also much interested in this question, and when they received the list of candidates from the medical board, they referred these names and the whole matter to a committee of three. This committee, after mature deliberation, reported their conclusions in the shape of the following resolution, which was unanimously adopted by the trustees (seven out of nine being present), on May 30, 1882:

"Resolved, that Dr. Sands be the sole attending surgeon for a period of one year, and that he have one assistant, to be appointed by the board."

Thus the Roosevelt Hospital assumed an advanced position in entrusting its entire surgical service to one head.

Is this extreme reaction from the former plan of broken services a desirable innovation? We doubt it.

It would seem desirable in a hospital to keep the door open to a wholesome competition, or rather emulation, between its higher medical officers. A single service, under one head, even though it be a most admirable one, can hardly help degenerating, or rather dropping into a stationary routine corresponding to the increasing conservatism of its chief. Besides, there are few surgeons who would not admit the advantage, in a practical and in a moral sense, of having at least one colleague of equal rank with whom to consult and with whom to vie in attaining perfection in professional work. The saying, "It is not good for man to be alone," applies, it seems to me, with much force to such a question. Yet, as compared with the former state of affairs, the present organization of the surgical service at the Roosevelt Hospital constitutes a great advance, and all who know the gentleman upon whom has fallen the responsibility of managing this whole service must feel that he, if any one could, will succeed by personal devotion and professional skill in conducting it advantageously to the patients and to the hospital.

Still, we cannot but hope that before long, as the hospital is enlarged, another surgeon will be appointed, and two continuous surgical services, each with a full resident staff, organized. One hundred beds would be sufficient for two quite active surgical services or divisions. The results to be obtained from such an ultimate and permanent arrangement would be collaboration and emulation between the two surgeons, the thorough education of a larger number of young surgeons in the house staffs, the giving of more individual care to each patient, the elaboration of bedside notes and histories of cases, and the more systematic use of the material of the services for oral teaching, and for publication. A little reflection and analysis will show to any reasonable mind that all these objects, though some of them at first sight seem to be in favor of the surgeons, or of pupils, in reality and practically tend to benefit the sick and wounded in the community at large. We are perfectly willing to admit that a hospital has for its first and chief and last object the care of the sick; but how much greater is the usefulness (in this sense) of a hospital that becomes a centre of instruction for its medical officers and for pupils from without, thus increasing and perfecting the medical skill which is to be available to the community, and even to the whole country.

One clause in the resolution by which the trustees of Roosevelt Hospital created a single surgical service—that relating to the appointment of an "assistant"—is much more important than appears on its face. If there were two surgeons of equal rank, with the understanding that both should never be absent (except a day or two) from the city at the same time, the use of the term "assistant," and the appointment to this position might hardly be worthy of comment.

Under the present circumstances the "assistant" will have to be the *alter ego* of Dr. Sands for several weeks probably, in charge of one of the most important surgical services in the country. He will have the care of many serious cases, will have to perform capital operations, will have the control, in a professional sense at any rate, of a staff of very intelligent *internes*, and yet he is expected to do all this without real rank, and, in the case of the present appointee, without sufficient professional standing. He is not Assistant Surgeon to Roosevelt Hospital, as he should be; and however talented and promising a man he may be (and we have good reasons for recognizing in the gentleman appointed the promise of all qualities necessary for the position), his name, his reputation, and his scientific contributions will not satisfy the demands of the profession and of the friends of Roosevelt Hospital when they ask who takes Dr. Sands' place; who has this splendid surgical service during Dr. Sands' unavoidable absence in the summer months?

We regret to say it, but we believe that in this is the chief weak point, if not a positively objectionable feature in the new departure at the Roosevelt Hospital. Through courtesy, and through a lack of appreciation of the true duties and responsibilities of the "assistant" named in the resolution of May 30th, the medical board and the trustees have made a mistake. The assistant surgeon of such a service should be almost if not quite the equal of its head in talent, experience, and years, because at any time the responsibility of the work may fall upon him, and because he is naturally a candidate for the position of surgeon should it become prematurely vacant.

Taking every thing into consideration, we think that the profession should look with hopeful satisfaction upon the experiment, and we trust that a progressive change in the same direction, though perhaps less extreme, will go forward in all our general hospitals. Some of them are on the good road, and will almost inevitably reach a result within the next four or five years.

Trustees or governors of hospitals, for the sake of the patients in whose behalf they administer their trusts, and medical men for the sake of humanity and of progress in medical science, should not cease asking for continuous services.

E. C. SEGUIN.

NEW BOOKS AND INSTRUMENTS.

RECENT AMERICAN WORKS ON OPHTHALMOLOGY.

(1) **A Treatise on Diseases of the Eye.** By HENRY D. NOYES, A.M., M.D., etc. New York: Wm. Wood & Co., 1881. (No. 12 of Wood's Library of Standard Medical Authors.)

(2) **The Diagnosis and Treatment of Diseases of the Eye.** By HENRY W. WILLIAMS, A.M., MD., etc. Boston: Houghton, Mifflin, & Co., 1881.

(3) **A Manual on Diseases of the Eye and Ear.** By W. F. MITTENDORF, M.D., etc. New York: G. P. Putnam's Sons, 1881.

(4) **A Manual of Ophthalmic Practice.** By HENRY S. SCHELL, M.D., etc. Philadelphia: D. G. Brinton.

It is curious, to say the least, that four books upon ophthalmology should have so suddenly been brought before the medical public from purely American sources, especially when we remember that until the appearance of the present volumes nothing more pretentious than Williams' small book on "Diseases of the Eye" could be reckoned as an American work on this subject. The efforts of our ophthalmologists seem to have been directed toward the editing of English works, as witness Bull's Am. ed. of Soelberg Wells, Green's ed. of Brudenell Carter, and the Am. ed. of Nettleship; or toward the translation of text-books from German and French sources, as in the Am. ed. of Stellwag, or the translation of Schweigger's "Hand-book of Ophthalmology." Of the four works at present under consideration, three at least claim positions as text-books; the last, in the words of the author, "only states briefly the generally accepted principles of ophthalmology," while "the small size of the book restricts allusions to authorities, to the history of the science, or to any other subject than the aforesaid principles and the treatment of the various conditions." It is a well-arranged, concise, practical manual, doing well what has frequently been done before, and making no pretensions to "filling a want long felt," etc.

The third upon the list, Mittendorf's manual, is rather pretentious for a manual, being of some 450 pages, and thoroughly equipped with ophthalmoscopic plates (from Liebreich and Wells), and plates showing diseased conditions of the membrana tympani from Politzer, as well as some illustrations from Sichel's Atlas, some of which are fairly well done, but others are execrable. The manual is a compilation, and from so comparatively young an author we ought not to expect any thing more. It evidences indefatigable industry on the part of its author, as well as a praiseworthy determination not to let his light be hid under a bushel. Its especial strength lies in the fact that it is double-barrelled, and in this respect the claim in the preface that it fills the long-felt want of a short, practical manual of the diseases of the eye and *ear* is valid, for, so far as the eye alone is concerned, the treatises of Williams, Lawson, Carter, the Am. ed. of Schweigger, *et al.*, have long since filled the gap. The combination of these two departments of practice (which are still so frequently, though so irrationally connected) in a single treatise, for the use of the economical student or country practitioner, is decidedly useful, and this, together with the judiciousness, thoroughness, and attention to detail which is everywhere apparent, has already carried the work to a second edition.

The main interest of the review centres around the first two works on the list. Their authors are representative men of different and rival cities, and though science is cosmopolitan we all know its practical application is apt to have local coloring. Both are men of large experience; one was, and the other is President of the American Ophthalmological Society, and their opinions upon mooted points of ophthalmology, of which there are not a few, have been looked for with interest by those who knew the books were in press. In general gracefulness of style, elegance of phraseology, and choice English, Williams' book has a decided advantage. Such innovations as "hit off the place" (Noyes, p. 72) are not found in it, while, on the other hand, it lacks the terseness of statement and systematic presentation of the different divisions of the subject which are so characteristic of all Noyes' scientific communications. It has been said that these books do not represent the American school of ophthalmology because there is no such school, which is very true, for American ophthalmologists are peculiarly cosmopolitan by education, drawing most largely from German sources, but also freely from the English and French ; and in these days of free intercommunication and international congresses, the different "schools" are separated by lines which are yearly growing less distinct.

Nor can these books be called "model" text-books, though either one is a fair representative. Williams' is better for the student who does not care to be encumbered with nice anatomical or pathological points or with frequent reference to the literature of the subject, while Noyes' is better for the more thorough painstaking man.

It is true that science is not advanced by text-books but rather by monographs ; nevertheless monographs reach but a few, and many who are reached do not comprehend their bearings or importance ; and it is absolutely necessary that every few years some text-book by a representative man shall gather together the grains of wheat which these monographs have brought along with their load of chaff, and put them into shape to be appreciated and utilized by the student and general practitioner. This is what has been done by the works now under consideration.

Each has its especial strong points : Noyes dwelling with more detail upon muscular derangements, plastic operations about the lids, and affections of the nerve ; while Williams gives more attention to superficial inflammations, as may be seen by his division of conjunctivitis into fifteen varieties against six by Noyes.

Noyes seems to have followed Schweigger and others in dividing his work into two distinct parts, of which the first embraces the different methods of examination of the eye, the *general* nature and treatment of ocular diseases, the errors of refraction and accommodation, and diseases of the muscles ; while the second part takes up the diseases of the different tissues in the anatomical order. Williams, on the other hand, after a brief consideration of methods of examination and of general treatment, begins with the conjunctiva and goes through the whole series of tissues systematically, and then, abandoning system, goes on with anomalies of refraction and accommodation, muscular disorders, ophthalmitis, orbital diseases, and lachrymal and palpebral disorders.

Noyes' chapters on the general anatomy and physiology of the eye, as well as on the blood-vessels of the globe, all of which are omitted from the other work, are very useful in leading up to the subject, and rounding out the work without adding essentially to its size.

In the chapters on general treatment we find both rejecting the use of leeches in reducing inflammation, and condemning blisters and setons as of little or no use. Indeed Williams calls the latter "a relic of barber surgery," which is rather strong language for a method advocated by Critchett, of London, as of great use in obstinate ulceration of the cornea.

The chapters on the ophthalmoscope are full and excellent in both works, but it is singular to find no allusion to the light streak on the retinal vessels in Williams' description of the ophthalmoscopic picture.

The statement in the same section, that "the direct method of examination is only exceptionally used for purposes of exploration to discover pathological changes," may hold good for the latitude of Boston ; but in New York it is, by most ophthalmologists, used in every case requiring ophthalmoscopic examination, and would be the one retained, if both could not be employed.

In the local treatment of phlyctenular conjunctivitis there is considerable discrepancy. Williams, rejecting calomel and atropine, relies upon pilocarpine (which remedy seems to have taken a strong hold upon his admiration) and very weak astringent collyria, while Noyes uses calomel, Pagenstecher's ointment, and in some cases atropine.

In gonorrhœal conjunctivitis Williams' treatment is not vigorous enough to meet the requirements of the New York plan. While agreed upon the advisability, in many cases, of canthotomy, there is a decided difference as to the subsequent course ; Williams relying upon cleanliness and very mild astringents, Noyes pursuing a more vigorous course, of leeches, cold compresses, and nitrate of silver.

In the treatment of *ulcer of the cornea* Williams makes a decided innovation in advising against the use of atropia, and justifies himself by saying : "For years I accepted this treatment by atropia, with suitable auxiliaries, as being the best within our knowledge, and a great improvement upon the more violent measures of the olden time. But I could never satisfy myself that any sedative action was exercised by atropia or other mydriatics in affections of the cornea. On the contrary, the increased sensitiveness to light, caused by the wide expansion of the pupil, seemed to more than counterbalance any possible benefit, because the spasmodic closure thus excited in the lids, and their close friction over the ulcer, could have only a painful and harmful effect, tending to extend and deepen the ulcer, to favor in some measure a forced infiltration of necrotic material from the ulcer between the layers of the cornea, and to increase the danger of a giving way of the cornea at its thin part and the protrusion of the iris through the perforation. This is no mere theory, but the conclusion reached after thoughtful and extended observation. The use of means calculated to intensify one of the most constant and characteristic symptoms of a disease, and in so doing to augment other morbid conditions, could scarcely be regarded as fulfilling the probable requirements for successful treatment. A substitution of remedies which relieved this urgent symptom appeared, therefore, to be a judicious as well as a scientific adaptation of means to an end, which was worthy of a practical trial. The discovery within a short time of the myotic qualities of Calabar bean and jaborandi, with their alkaloids, eserine and pilocarpine, by means of which we obtain the long-wished-for power of contracting the pupil at will, has placed at our disposal two invaluable remedies. My experience in careful trials of these myotics in corneal affections has been confirmed by that of other observers in Germany and elsewhere, some of them speaking of these as the remedies of the future in corneal affections ; and it seems to be demonstrated that the advantages hoped for from their use are to be realized."

In the treatment of opacities of the cornea Williams advises against the use of local stimulants, believing that the improvement sometimes seen is a *post hoc*, and not a *propter hoc*. Noyes advises local stimulation in non-vascular or slightly vascular ulcerations.

As regards arresting staphyloma of the cornea by iridectomy, Williams is an unbeliever, saying it is of no avail. Noyes holds that in some cases a broad iridectomy will " reduce intra-ocular tension and confer a measure of sight."

The treatment of pannus by inoculation is regarded by Williams as unjustifiable, but from the way in which he speaks of it, it would seem that he has had no experience with it ; while Noyes, who speaks from the vantage-ground of some successful cases, thinks that in cases of complete pannus in vigorous subjects the cornea may sometimes recover its clearness.

The removal of pterygium is conceded by both to be best effected by excision, but while Williams advises against any attempt to cover the raw surface of the sclera by conjunctival flaps, as militating against the certainty of cure, Noyes gives directions for making the flaps, which do not differ essentially from those advocated by Knapp some years ago. Williams' position, that "all preparations of lead should be stricken from the list of remedies which may be used in the eye," seems to us the only safe position for a text-book to take; and the statement, that "usage gives currency and they may sometimes be used without harm," given in the other work, is weak and unworthy of the book.

The very important subject of iritis discloses little discrepancy of opinion except in the treatment of serous iritis, which Noyes does not make essentially different from that of the plastic form, while Williams says of it that "atropia is by no means to be regarded as the treatment, *par excellence*, to be used early and often as in the plastic variety." The latter considers a solution of the strength of two grains atropiæ sulph. to ℥ i of water sufficiently strong to rupture adhesions; while the former advises at least double that strength, and in New York even eight grains to the ounce is frequently used.

The chapters on cataract have an especial interest by reason of the debatable points upon which we are constantly seeking the results arrived at by our most experienced ophthalmologists.

Noyes would operate on *traumatic* cataract in one eye because of "gain in enlarging the field of vision, in the stronger mental impression, and because, notwithstanding no correcting glass was worn, a degree of stereoscopic vision was secured which the patients many times found of great advantage." Williams would not operate "because the eye operated on, having no accommodation, does not harmonize with the more perfect visual act in the opposite eye, and at times confuses instead of aiding it."

The latter would operate on both eyes at once in senile cataract, if both were ready; the former would not, preferring to give the second eye the advantage gained from the experience of the first operation. Anæsthesia by sulphuric ether is recommended in most cases by Williams with a fervor inspired by the sight of the statue in the public gardens commemorating an event alluded to by all Bostonians who write medical books. Noyes says the employment of anæsthesia "depends on the habit of the operator and the wishes of the patient." The use of corneal sutures to close the wound after extraction of the lens, which was first proposed about ten years ago by Williams, is still recommended by him, though it is certainly not in general use among ophthalmologists. Removal of soft lens matter by aspiration is condemned by both authorities.

As regards the best method of extraction for the majority of cases, we find Williams rather non-committal. According to Noyes, the modern operation known as Graefe's modified linear extraction has quite supplanted the "flap method," while Williams evidently has a fond affection for the "flap method," which he performed so skilfully long before the new method was devised, and he draws attention to the fact that the development of secondary consequences—viz., separation of the retina, and irido-cyclitis—is beginning to be noticed in an alarmingly large number of cases.

The new operation of optico-ciliary neurotomy as a substitute for enucleation is more enthusiastically mentioned by Williams, who says, "there appears to be every reason for believing that it may replace the graver operation (enucleation) in a large number of the cases, at least, where enucleation has been performed as a preventive measure, before the second eye has become affected." Noyes says, "it has claims to consideration and is being extensively tested."

Of sclerotomy, the proposed substitute for iridectomy in glaucoma, Williams says: "It seems to be conceded that though sclerotomy offers a fair chance of success, iridectomy is most to be relied on for a certain and permanent curative effect, and it is, perhaps, sometimes useful for the relief of persistent pain in absolute glaucoma, where vision is hopelessly lost." Noyes says: "My own experience with it is small, and leaves me to favor iridectomy in critical cases. The efficacy of the proceeding is not fully established, although some have written strongly in its favor."

In the treatment of stricture of the lachrymal passage there is a decided difference of opinion, Noyes favoring thorough incision at once, followed by the use of *large* probes, even as large as 4 mm. in some cases. Williams, on the contrary, incises very cautiously, and uses probes of moderate size only.

The subject of color-blindness is lightly touched upon by Noyes, who does not seem to have taken any special interest in the investigations which have lately been made in this department, if one may judge from such inaccuracies as saying in Holmgren's test that the patient is requested to match *shades* of color, or saying that the proportion of women who are color-blind is 2 per cent. instead of $\frac{1}{4}$ of one per cent. Williams' chapter on the subject is full and accurate, except that he *understates* the proportion of the defect in females, placing it at $\frac{1}{40}$ of one per cent.

It is unfortunate that so excellent a work as that of Noyes should come to us in so mean a garb. The paper is poor, the wood-cuts in many cases miserably done, and in short the whole execution is unworthy of the subject-matter, if we except the ophthalmoscopic plates, which are better than those in the Boston book, which, coming from the Riverside press, fully sustains the high reputation of its predecessors, and its perusal is not calculated to increase any of the disorders of which it treats. [S. B. ST. J.]

The Physician Himself and What He Should Add to the Strictly Scientific. By H. W. CATHELL, M.D., Late Professor of Pathology in the College of Physicians and Surgeons of Baltimore, Ex-President of the Medical and Surgical Society, Active Member of the Medical and Chirurgical Faculty of Maryland, Honorary Member of the Lincoln Philosophical Society, etc., etc. Baltimore: Cushings & Bailey, 1882, pp. 194.

"Professional tact and business sagacity," says the author of this valuable little work, "are as necessary to the physician as the mariner's compass is to the navigator. There are gentlemen in the ranks of our profession who are perfectly acquainted with the scientific aspects of medicine, and can tell you what to do for almost every ailment that afflicts humanity, who, nevertheless, after earnest trial, have never gotten either reputation or practice, because they lack professional tact and business sagacity; and there is nothing more pitiful than to see a worthy physician deficient in these qualities, waiting year after year for practice that never comes. * * * You will find that intellect, genius, temperance, correct personal habits, and other excellent qualities will all fail unless you add ambition, self-reliance, and aggressiveness to them." The bock is in fact a mine of practical hints, commonplace it is true, yet valuable because they are commonplace. To the young practitioner it will prove a guide to correct business habits in many ways, and will put him upon his guard against multitudes of little obstacles that beset the pathway to success. To those who have been longer in the profession it will serve as a prompter to re-establish methods that have fallen into disuse from lack of time, negligence, and indifference. No one can read the book without finding some point which possesses a personal application impelling to more caution, or more vigorous action, as the case may be. We cannot repeat sound maxims too often, and though we may differ in opinion with the author in many particulars, such differences of themselves set us thinking upon details that are too frequently neglected. The difficulties of writing such a work can hardly be overestimated, and the author is to be congratulated in having considered such a multitude of subjects in a manner above criticism; on the other hand, the varied circumstances of the medical practitioner's life make certain of the author's maxims which may be applicable to one man, impracticable, or even ridiculous, for another. The intelligent reader will make allowance for this; but then comes another class of statements, too numerous to consider in detail, which must meet with solid opposition from a practical or an ethical standpoint, and which by some will be considered not merely conservative but old-fogyish. The general tone of the work, however, indicates a bold conservatism, a happy blending of egoistic and altruistic notions. The author's style is terse, fearless, at times humorous.

Occasionally suggestions respecting treatment are introduced which seem a little foreign to the plan of the work. The following quotation is, to say the least, quaint: "Remember that the act of nursing not only supplies the babe with nourishment, but also communicates the mother's heat and electricity; it is therefore well to have all *hand-fed babes* nestled to some one's bare breast at intervals of a few hours, in exact imitation of those that suck." A few other quotations will serve as better examples of the abundance of common-sense which pervades the volume, and the bold, clear style in which it is written. "When you hear of a doctor who wishes to be considered especially clear, or ahead of others, or extraordinarily fair in his opinion, boasting that he 'does not believe in drugs,' 'depends on nature,' etc., you can safely conclude that in his zeal to become a medical philosopher, he has doubtless lapsed in his materia medica, or overstated his credulity, or that his usefulness has run to seed."

"You are bound, as if by an oath, to use your best judgment for every one who puts himself under your care, but neither the code of ethics nor the code of honor prevents you from sailing as near to every popular breeze as truth and justice will allow."

"You will not have practised long before you will find that your welfare will not depend upon how much you book, but upon how much you collect, and that if you never insist upon the payment of your fees you never can separate the chaff from the wheat."

"Flexibility of manner, self-command, quick discernment, address, and ability to adapt yourself to the ever-changing phases of medical practice, are great necessities; the lady in her boudoir and the hod-carrier, the beer-seller and the clergyman, the aged and the young, should each find in you his ideal:—seek to become perfect in this power of adjusting yourself."

The work is dedicated to Prof. Austin Flint, Sr., "in admiration of his various contributions to scientific medicine, and of his untiring devotion to the welfare of our profession." [W. R. B.]

ORIGINAL OBSERVATIONS.

A CASE OF BULBAR APOPLEXY; BILATERAL FACIAL PARALYSIS; PARTIAL PARALYSIS OF THE TRIGEMINUS; HEMIPLEGIA.

By HENRY J. BERKLEY, M.D.,
Baltimore, Md.

The patient, Mary B., æt. 40, a stout and vigorous, though intemperate woman, was stricken by paralysis in June, 1881.

Headache, severe and continued, was complained of for several days before the occurrence of the lesion.

When asked to mark out its locality, she lays her hand over the posterior region of the skull.

I requested the woman to describe to me how the seizure began. She answered: "I was standing upright when suddenly I felt drunk without having taken any liquor"; in other words, her head swam, she felt dizzy, staggered, and fell.

As soon as she had somewhat recovered consciousness she perceived that the right side of the body was incapable of motion; that she was paralyzed.

At the same time, she also remarked the hearing of the left ear had suddenly become bad.

A few days after the stroke she noticed that the eyesight of the left eye was growing dim; soon the cornea became milky, gradually increasing until total blindness followed.

She tells me that she was completely paralyzed shortly after the attack, but that she never lost the feeling on either side of the body.

Stiffness of the paralyzed limbs commenced some weeks after the apoplexy, lasting between two and three months.

There has never been any embarrassment of respiration.

Physical Examination.—When the patient first came under my care, some four months after the attack, I found the right side paretic ; she could move the right leg slightly, and the arm could easily be raised to the face, only, however, with a very ataxic movement ; if I asked her to close the eyes, and touch the tip of her nose with the right index finger, she would invariably miss it, either putting the finger into the eye or upon the forehead.

The motions of the left hand and arm were natural.

On testing the strength, I found great loss of power in the right hand.

There was bilateral facial palsy, involving both superior and inferior facial muscles ; the features were stiff and immobile, the cheeks drooping, complete closure of the lips could not be effected, whistling was impossible, and the pronunciation of the labials *b*, *p*, *v* was incomplete. This facial condition (paralysis) was much more marked than that of the body.

The tongue is protruded to the right with an irregular, uncertain motion ; its upper surface is concave when put forth.

No atrophy is present in any of the muscles ; their faradic contractility is well preserved.

The facial muscles of both sides respond to faradism.

For several months after the date of the seizure (about five) there was a tonic spasm of the jaw muscles (temporal, masseter, etc.), so that it was impossible for the patient to open the teeth more than the one fourth of an inch.

Never has there been any dysphagia. The nails of both feet and hands are not in the slightest degree ridged; on the paralyzed side they are observed to grow faster than on the sound.

There is incontinence both of urine and of fæces.

Tactile sensibility on the hemiplegic side is good everywhere, with the exception of a small portion of skin about the middle third of the forearm, on its flexor surface; in this spot there was also slight analgesia.

On the right face sensibility is preserved, with likewise the exception of a little patch anæsthetic over the lower portion of the masseter and buccinator muscles.

On the left half of the face the whole of the region supplied by the supra-orbital branch of the trigeminus is totally analgesic and anæsthetic ; nowhere else is there the slightest diminution of sensation.

Tactile sensibility on both sides of the tongue is good.

The special senses were very carefully examined.

Taste and smell were intact ; bitters, salts, and aromatics being recognized without difficulty.

Auditory examination, by means of a watch, showed hearing to be totally extinct in the left ear, both by contact with the cranial bones and through the medium of the air, while in the right the function was normal,

Speech is slow, jerky, and drawling, but there is no aphasia.

The left eye presents an exceedingly interesting condition ; as before mentioned, shortly after the apoplectic stroke, the cornea became cloudy, thickened, and fleshy. At the time I first saw it the pannus protruded more than the one fourth of an inch beyond the lids, which were separated by the growing mass.

Apparently the protrusion of the eyeball is entirely due to the hypertrophied cornea, for on measuring the two sclera as near as possible to the corneal margins and comparing, there seems to be no appreciable difference in their level.

Contrary to the usual history, the cornea did not rupture.

During the last three months of life the cornea atrophied, the vascular injection lessened, and before death its apex sank below the level of the sclero-corneal margin.

Sensation in the diseased cornea is almost entirely abolished.

There is increased secretion of lachrymal fluid, the eye weeping continuously.

The movements of all the ocular muscles are perfect, nor is there any ptosis.

About the same period that the hypertrophied cornea began to subside, the spasm of the left jaw muscles, together with the paralyzed right facial muscles, ameliorated greatly, the woman regaining almost perfect control over the right face, and was enabled to open the mouth nearly to its full extent.

At times flushes of redness and heat, accompanied by slight tingling, will pass over the limbs and trunk of the right side ; indeed, the whole hemiplegic side has a more reddened hue than the sound.

As far as I can judge there is no loss of intellectual power ; the woman herself thinks her memory to be quite as good as ever it was.

There is some emotional disturbance, the patient laughing or crying at words or acts that would not be noticed by a healthy person.

The urine was only examined once ; it contained no albumen, nor has there ever been any polyuria.

Some two weeks before the end, bed-sores formed for the first time during her illness, commencing upon the small of the back, then over the sacrum. At the time of death the integument had sloughed to a considerable extent.

Etiology.—Although undoubtedly a woman of loose morals, no symptoms of syphilis could be detected on the most patient examination ; nor was there any abnormality in the heart's sounds, the rhythm being perfectly regular. Indeed, the only probable cause that could be obtained was that she had been a hard drinker, and I think that alcohol, most probably, was the cause of the arteritis that led to the formation of miliary aneurisms, the rupture of one of them giving rise to the hemorrhagic *foyer*.

Death, May 21, 1882, from gradual asthenia.

A diagnosis of hemorrhage into the bulb was made by Professor F. T. Miles, of Baltimore.

I owe the privilege of an autopsy to Dr. H. Woods, of Baltimore, the case having passed into his hands before death.

Necropsy, 28 hours after death.—No measurable difference could be found in the size of the extremities when compared.

The eye was completely atrophied, the cornea sunk, the sclera thinned ; even the lens had been absorbed.

The heart was small and very fatty ; the valves, aortic and mitral, were perfect in their junction, but were, however, very slightly thickened, with here and there an atheromatous patch, the degeneration not being sufficient to cause a murmur during life ; atheroma was also seen in the aorta.

In the abdomen the liver and kidneys were considerably diseased.

The brain : The skull was of great thickness ; the diplöe large, its meshes open, constituting by itself the greater part of the bone.

The dura mater was of normal aspect, not adherent to the cranial bones, nor to the meninges.

The lateral sinuses contained dark fluid blood.

The arachnoid presented that peculiar thickening so often found in atrophied brains (alcoholism).

The pia mater was opaque, but not adherent to the cortex.

Between one and two ounces of clear serum had collected in the arachnoid space, mostly at the base.

The vessels, basilar and carotids, were strewn with hard, white plaques of atheroma ; in several places the calibre of the arteries was very considerably reduced in size.

On the basilar artery, at the inferior margin of the pons Varolii, a fusiform aneurism of the size of a bean was found.

The seventh pair of the left side was seen to be slightly smaller and of softer consistence than the nerve on the opposite side. No departure from the normal was found in the optic nerves.

The hemispheres were atrophied considerably, not nearly filling the cranial cavity.

The convolutions were of simple arrangement, and seemed slightly flattened; the sulci did not attain any great depth between the convolutions.

The brain tissue was very soft, more than could be accounted for by *post-mortem* changes, but there was no localized degeneration of any kind on the cortex. Section of the white and gray substance revealed nothing abnormal ; the ganglia exhibited their usual gray color; the internal capsule ran its regular course; everywhere the different parts were normal.

The lateral ventricles were dilated, their ependyma thickened ; both ventricles contained a quantity of serum.

The aqueduct of Sylvius was slightly dilated.

The cerebellum was injected, probably *post mortem*, but was otherwise normal.

The bulb : On the floor of the 4th ventricle, on the left half, just above and to the outer side of the eminentia teres, was a small depression of irregular shape, slightly yellowish in color.

Transverse section disclosed an old hemorrhagic focus, of stellate form, extending from a point 5 mm. above the lower margin of the pons, 5 mm. internally to the floor of the ventricle, reaching upward nearly to the superior border of the pons, progressively growing smaller as it ascended. The centre of the lesion lay anterior to the genu nervi facialis. From about this point two arms extended from the focus far forward, nearly reaching to the border of the superficial transverse fibres of the pons. Nowhere did it approach the median line. Only at the point already mentioned did the focus touch the floor of the ventricle.

In the right half of the bulb, at a point slightly anterior to the nucleus of the facial nerve, was another focus of the size of a No. 5 bird-shot, yellowish in color, and apparently about the same age as the larger lesion.

Microscopic examination.—On examination of the smaller arteries, particularly those of the anterior perforated space, I found several miliary aneurisms.

The walls of the vessels showed the lesions of chronic arteritis.

Sections of the hardened cord, stained with carmine, showed a descending degeneration of the crossed pyramidal tract of the right half of the organ, and a corresponding sclerosis of the direct tract on the left.

The tissue was less densely sclerosed than is found in old cases of absolute paralysis, many more of the axes-cylinder seemingly being intact.

Apoplexy of the medulla, in which the patient survives for any length of time, is of comparatively rare occurrence. In a table collected by Gintrac[1] of 560 cases of cerebral hemorrhage in which there was a single clot, 76 were in the pons Varolii and crura cerebri, while only two were in the medulla proper. Hemorrhage is very frequent in aneurisms of the basilar artery and its branches. According to Lebert (*Berl. klin. Wochr.*, 1866): "Among 31 cases of aneurisms of the base, vascular ruptures and hemorrhages occurred 17 times into the parenchyma of the pons and medulla oblongata."

Fig. 1.

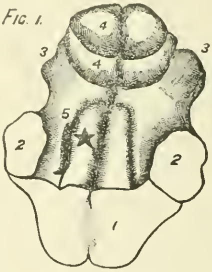

Fig. 2.

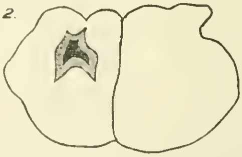

FIG. 1.—Oblique view of medulla and pons; 1, transverse section of surface of medulla; 2, transverse section of cerebellar peduncles; 3, cut crura cerebri; 4, corpora quadrigemina; 5, cicatricial depression in floor of fourth ventricle, immediately over the hemorrhage, marked by *

FIG. 2.—Transverse section of the pons just below exit of fifth nerve, showing the site of the hemorrhage within the pons.

[These diagrams have been drawn from the specimens. The reader who is specially interested in studying the localization and relation of the lesion more minutely can do so by projecting the site of the hemorrhage upon the various plates and diagrams of the medulla oblongata in Ziemssen's Cyclopædia, Am. ed., vol. xiii, pp. 838–844.—EDITOR.]

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[1] Cited by Hammond, "Diseases of Nervous System."

Echeverria records a case of aneurism of both vertebrals, near their junction to form the basilar; the tumor on the right compressed the roots of glosso-pharyngeal and hypoglossus, which (tumor) occasioned during life dysphagia and paralysis of the tongue. Death sudden from hæmatorrachis.

During the past winter I met with another aneurism of the basilar, in about the same locality as in the present case; the artery also presented the anomaly of being formed by the junction of the vertebrals at the inferior border of the foramen magnum. Death resulted in this case from rupture of the external arteries of the lenticular ganglion.

The most difficult symptom to be understood in this case is the bilateral facial paralysis. To account for it we shall have to accept one of two hypotheses: first, that the second lesion in the right side of the medulla caused it; at the beginning having an additional influence by pressure upon the remaining sound portions of the nucleus, and when, in the course of time, by gradual absorption, permitted more complete motor neurization of the remaining intact fibres of the facial, then the muscles partially regained their former power. Otherwise, we must follow the descriptions of Bastian and Hammond, viz.: "that injuries of the upper part of one lateral region of the pons produce a hemiplegia of the opposite side of the body, accompanied by facial paralysis of the same side as the hemiplegia." In other words, if the lesion happen above the decussation of the encephalic fibres, the paralysis will be on the opposite side for all parts of the body.

Gowers, in the January issue of *Brain*, of 1880, mentions the frequency of bilateral symptoms of paralysis of the cranial nerves in the diseases of the pons Varolii.

Following Vulpian (*Essai sur l'origine de plusieurs paires de nerfs craniens*), the decussation of the roots of the facial "occurs in the mesial line of the medulla oblongata, at the junction of the two nuclei of origin."

In the present subject the long axis of the lesion ran in the long diameter of the bulb, and possibly, in addition to destroying the lower nuclei, it may also have intersected the fibres coming from the cerebrum.

The partial involvement in paralysis of parts of the motor and sensory branches of the trigeminus is one of the most important circumstances of the case, and may be a valuable addition to localization in the anatomo-pathology of the bulb.

But first to recapitulate: There was absolute anæsthesia and analgesia of the supra-orbital division of the ophthalmic; perfect sensibility in the superior maxillary, malar, and mental branches; and a tonic spasm, lasting several months, of those muscles, principally the masseter and temporal, supplied by the motor division of the fifth; all on the left side, the right not participating.

I do not think it would be carrying a theory too far, to say that here, not judging from mere hypotheses, but from actual examination of the locality of the hemorrhage, drawing my authority for the location of the nuclei from Erb, we have destruction of the superior sensitive, together with compression of the motor nucleus of the fifth.

Now, from this fact, it is a very plausible hypothesis to consider that this superior nucleus is the spot from which alone are derived the fibres that convey sensation from that part of the face, corresponding to the distribution of the terminal filaments of the ophthalmic branch of the trigeminus, and that the inferior and larger nucleus supplies the fibres to the inferior regions of the face. The non-participation of this latter nerve centre can only be explained by the supposition that the inferior nucleus lies more in the postero-lateral portion of the bulb than it is usually considered to be, that portion not having been destroyed for 5 mm. from its outer margin.

Regarding the trophic disturbances following the destruction of the ophthalmic branch of the trigeminus, there is yet no definite settlement of the question as to whether the result is from the lesion of the nerve or from mechanical irritation of the exposed cornea.

The numerous experiments of Snellen, Meissner, Schiff, and others all lead to different results.

Samuel,[1] "after having produced a violent irritation of the Gasserian ganglion in rabbits by means of the induced current, observed the development of acute ophthalmia, accompanied by hyperæsthesia of the eye."

"In rabbits in whom the trigeminus has been cut within the cranial cavity, ulcerations readily occur upon the nose, lips, palate, and sometimes upon the tongue." (Rosenthal.)

These parts being habitually exposed to irritation, it cannot be deduced in favor of the theory that the keratitis is due to mechanical lesion of the exposed surface.

Samuel (Die trophischen Nerven, 1860), cited by Rosenthal. Diseases of the Nervous System. American edition.

Bock and Friedrich[1] have seen "cases in which ophthalmia developed in persons (suffering from tumor) despite the preservation of sensibility."

Weir Mitchell records a case (injuries to nerves) of a gun-shot wound of the ophthalmic branch of the fifth, in which, in less than three days from the time of the injury, the cornea became "dull, insensible, and slightly clouded." Surely this is not time for any external irritation to produce such results.

According to Charcot and Friedrich,[2] "the trophic disturbances of the eye are caused by an inflammatory irritation (descending neuritis)."

"In an experiment performed by Buettner,[3] after multiple incisions into the Gasserian ganglion, and a purulent ophthalmia of rapid course (despite the application of protecting apparatus), the ganglion was found in a condition of traumatic inflammation, extending into the ophthalmic branch."

On the first examination of the focus it is difficult to conceive why the nucleus of the abducens was not destroyed. By more careful investigation the hemorrhage is found not to extend within 3 mm. of the eminentia teres, immediately within which lies the nucleus of the abducens (Erb), while at a greater depth, coming within the limits of the lesion, is the nucleus of the facialis, which was totally destroyed.

Scrutiny of the lesion revealed all that tract of the medulla anterior to the striæ acusticæ, destroyed, involving the "inner" and "outer" nuclei of the acoustic of Huguenin.[4] This destruction I believe fully sufficient to cause the total auditory anæsthesia, even though the remaining nuclei of the acoustic were not involved.

There can be little doubt that the vaso-motor centres were injured, perhaps primarily in the medulla, then secondarily in the lateral columns and gray horns of the cord. The increased redness, vascular dilatation, etc., of the hemiplegic side showed that the vaso-motor contraction of the arteries was destroyed or lessened.

Owsjannikow and Dittmar have defined the vaso-motor centre in the medulla of the rabbit "as lying between the corpora quadrigemina and calamus scriptorius, somewhat removed from the middle line, in the lateral columnar tract of the formatio reticularis.

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[1] Bock and Friedrich, cited by Rosenthal, *l. c.*, p. 485.

[2] Charcot and Friedrich, cited by Rosenthal, *l. c.*, p. 485.

[3] Buettner, Rosenthal, *l. c.*

[4] Huguenin, "Allgemeine Pathologie der Krankheiten des Nervensystems."

"The nerves for vaso-motor contraction which leave it are said to run through the lateral columns to the anterior nerve roots." (Dittmar.[1])

Even in man this demonstration would account for the vaso-motor lesions in this subject, for the tract destroyed by the hemorrhage corresponds to the location described by Dittmar in the rabbit.

The hemiparesis was owing to the circumstance that an arm from the focus intersects the pyramidal tract at its extreme outer edge, as can be readily seen in several of the sections, especially those of the middle portion of the pons.

Rigidity of the paralyzed limbs is very frequently encountered in bulbar apoplexies. (Bastian.)

It is a question whether the anæsthetic spots on the right side, on the arm and face, have any significance whatever; they did not correspond to the distribution of any special nerve, and more probably belong to some mere local cause, such spots being not infrequently met with when the cutaneous surfaces are examined with care.

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[1] Owsjannikow and Dittmar, cited by Erb, Ziemssen's Cyclopædia, vol. xiii, p. 861.

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ARCHIVES OF MEDICINE.

Original Articles.

TUBERCULAR TUMORS OF THE WINDPIPE. TUBERCULOSIS OF THE LARYNGEAL MUSCLES. A CONTRIBUTION TO THE PATHOLOGICAL HISTOLOGY OF LARYNGO-TRACHEAL PHTHISIS.

BY JOHN N. MACKENZIE, M.D.,
BALTIMORE.

I.—TUBERCULAR TUMORS OF THE WINDPIPE.

THE larynx of the consumptive is the seat of various forms of neoplasm which are pathologically separable into three distinct groups.

1. *Granular hyperplasiæ.*—This is the most common variety of outgrowth, and consists of an aggregation of small grayish or pinkish granulations, which fringe the edges or deck the base of tubercular ulceration. They are rarely, if ever, met with as the result of the simple catarrhal or diphtheritic[1] changes which are found in phthisis of the windpipe, and are therefore of considerable value in differential macroscopic diagnosis. They occur in all portions of the larynx, their seat depending on the location of the ulcerative process, and are sometimes so abundant as to cause stenosis and necessitate tracheotomy. Anatomically allied to granulation tissue, they may be regarded as the representatives of a conservative process—*as a natural step*

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[1] On diphtheritic ulceration, see papers by the author in the *Monatsschrift für Ohrenheilkunde*, No. 9, Berlin, 1881, and the "Transactions of the Med.-Chir. Faculty of Maryland," 1882.

toward cicatrization. If sections be made through a tubercular ulcer of the larynx or trachea, and its surrounding granulations, a more or less clearly defined zone of hyperplastic inflammation of the connective tissue will be discovered, which isolates the degenerate tubercle from the healthy tissues in its immediate vicinity. The vegetations under discussion are the result of this protective inflammatory process, and consist, histologically, of a mass of newly formed connective-tissue cells and nuclei, in which enlarged, tortuous capillary vessels are sometimes developed.

2. *Papillomatous excrescences.*—Of less common occurrence than the preceding, the members of the second group are closely allied to simple laryngeal papillomata, and, like them, owe their origin to chronic vascular congestion of the mucous membrane. Their occasional presence, however, in the midst of pronounced pallor of the larynx, would lead to the belief that they sometimes result from the irritation of an opposite pathological condition, viz.: the peculiar anæmia of the laryngeal membrane, which is so often the herald of the tubercular process. Warty, acuminate, or leaf-like in form, and varying in color from a pale grayish to a pronounced reddish hue, they spring, as a rule, from the posterior laryngeal wall, and in this situation sometimes attain to a considerable size, and are readily mistaken for true laryngeal papillomata. They occur also on the epiglottis and below the anterior commissure of the vocal cords. In structure they are the histological analogues of the laryngeal papilloma, and occur, as a rule, independently of well-marked tubercular lesions. These growths often ulcerate and assume an apparently malignant form, but I am unaware of any microscopical evidence of their conversion into tubercular products. Stoerk,[1] who has carefully studied the development of these neoplasms, maintains

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[1] *Klinik d. Krankheiten des Kehlkopfes*, etc., Stuttgart, 1880, S. 282.

that their presence in the interarytenoid fold is an infallible sign of incipient tuberculosis, and, following Rokitansky, regards them as the result of the indurative proliferation of the connective tissue, which occurs in the course of chronic tubercular disease of the mucous membrane in the neighborhood of the arytenoid cartilages.

3. *Tubercular tumors.*—The two above-described varieties of neoplasm contain no histological elements by which they may be differentiated from simple granulation or papillary vegetations. The third group, on the other hand, includes solitary tumors of the windpipe, which are composed, histologically, of a mass of closely aggregated miliary tubercular nodules, and which occur independently of infiltration and ulceration of the mucous membrane. I desire to call especial attention to these growths, as they are not described in works on laryngeal pathology, and I am unaware of a single recorded case of laryngeal neoplasm whose tubercular nature has been demonstrated by the microscope.[1] The two following, which came accidentally under my observation in the Rudolph Hospital in Vienna, are therefore worthy of publication. The *post-mortem* examinations were made by Dr. Hans Chiari, to whom I am indebted for the specimens, and the organs handed to me for microscopic investigation.

1. *Tubercular tumor of the trachea.*—This was taken from the body of a man who had died in the hospital from carcinoma of the stomach. Secondary cancerous deposits were present in the liver, kidneys, spleen, and other organs. The lungs, however, contained tubercular cavities. The pharynx, larynx, and trachea were free from inflammation and ulceration. The bronchial and retro-tracheal glands

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[1] It is obvious that neoplasms of various kinds may be found in the air-passages of phthisical persons, which have no connection whatsoever with the tubercular process. The cases recorded by Andral *Clinique médicale*, t. ii, p. 103, (and *Spillan's Transl.*, Lond., 1836, p. 489), Trousseau "Laryngeal Phthisis," Phila., 1839, p. 31, and others, evidently belong to this category.

were enlarged, tumefied, and caseous. In the membranous posterior wall, at its junction with the cartilaginous framework of the trachea, about 1½ cm. above the bifurcation, was a well-defined circumscribed tumor about the size of a small bean, its long axis parallel with that of the trachea, and of a uniformly even, smooth appearance. It was covered by the mucous membrane of the trachea, and was dense in consistence, giving to the touch the sensation of a hard cancerous nodule, for which, indeed, it was mistaken. A similar growth was found in the pericardium.

The microscope revealed a picture for which I was not prepared. It showed, namely, that the tumor, which seemed to have its origin in the submucous connective tissue, consisted, in the main, of an aggregation of distinct tubercular nodules set in a more or less well-marked vascular net-work of hypertrophied connective tissue. The majority of the tubercles lay in the deeper portions of the mucous membrane and in the submucous tissue; a few were more superficial, lying under the epithelium. They exhibited all grades of degenerative change; in some caseation was so far advanced that nothing remained but the cellular wall. Between the individual nodules the connective tissue was hypertrophied and the seat of a moderate amount of round-cell infiltration which had invaded the glandular follicles in its vicinity. The tissues of the trachea in the immediate neighborhood of the growth presented no remarkable change. *The nodule in the pericardium showed the same histological structure that was found in the tracheal neoplasm.* All the other diseased organs were shown by the microscope to be cancerous.

2. *Tubercular growth of the vestibulum laryngis.*—The subject from whom this specimen was taken died outside of the hospital, of pulmonary phthisis. The whole upper compartment of the larynx, including the epiglottis, ary-

epiglottic folds and ventricular bands, presented a remarkable mammillary or granular appearance, due to the presence of small, uniformly smooth, dense, moderately hard nodular growths, which lay beneath the mucous membrane. The nodules were about the size of a split pea, each merging into its neighbors, so as to form one continuous growth. This process ceased abruptly on either side, at the free border of the ventricular band. There was no trace of ulceration in pharynx, larynx, or trachea.

Microscopical examination of numerous sections of the growth showed it to be of the same nature as the above-described neoplasm in the trachea. Like it, it seemed to have invaded principally the submucous tissue, and, like it, consisted of a mass of closely aggregated miliary tubercles in all stages of degeneration. The nodules in the deeper portions were further advanced in caseation than those which were more superficial. The growth was less dense in consistence than the one observed in the trachea, and the connective tissue between the nodules had undergone a less degree of hypertrophic change. The tubercles lay in close proximity to the blood-vessels, one or two of which were compressed by the new formation. The remaining portions of the larynx were healthy. The lungs contained tubercular cavities. There were tubercular ulcers in the intestine, and the basilar surface of the brain was covered with miliary granulations.

As intimated above, these cases are probably unique. Such tumors doubtless have a similar origin to the so-called " metastases " in the laryngeal mucous membrane which take their departure from old tubercular disease of other organs, as the kidney (Köhnhorn[1]) and bronchial glands (Lennox Browne[2]).

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[1] *Berliner klin. Woch.*, 1876, Nos. 3 and 4.

[2] *Med. Times & Gaz.*, 1876, i, p. 456. (Eppinger: " Pathologische Anatomie des Larynx u. der Trachea." Berlin, 1880, S. 155.)

II.—TUBERCULOSIS OF THE LARYNGEAL MUSCLES.

The hoarseness without palpable *intra-vitam* or *post-mortem* changes in the larynx, so often met with in phthisical patients, led Fraenkel,[1] in 1877, to examine the laryngeal muscles as a possible seat of degenerative processes which might explain the alterations in the voice. In twelve larynges, some apparently healthy, others presenting ulceration, he found constant anatomical changes, which consisted essentially in an atrophy of the striated muscles. The connective tissue between the primitive bundles was markedly infiltrated with cells, and the nuclei of the muscles so increased as to destroy apparently the muscular fibres by pressure of the new-formed cells.

Several years later, Posadsky,[2] of St. Petersburg, confirmed the observations of Fraenkel, and showed that the same degenerative changes took place also in the muscles of the upper and lower extremities, the intercostals, and the diaphragm. The muscles were pale and friable, and the microscope showed granular degeneration of the muscular fibres. In many cases there was disappearance of the transverse striation, and the fibres were notably narrowed, and some empty sarcolemma sheaths were seen. The interstitial connective-tissue changes described by Fraenkel were not discovered by Posadsky.

Heinze,[3] who has carefully studied the pathological anatomy of laryngeal phthisis, is unable to confirm the results arrived at by Fraenkel, and thinks that tubercular infiltration of the tissues is sufficient to explain the anomaly in the voice. This observer has furthermore discovered tubercle in the muscular tissue of the deeper laryngeal structures, an occurrence, according to him, of extreme rarity. Out of fifty carefully examined cases, tubercle of the muscles, unconnected with general tubercular infiltration of the larynx, was found only twice.

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[1] "Ueber die patholog. Veraenderungen d. Kehlkopfmusculatur bei Phthisikern." *Virchow's Archiv*, lxxi, 3, 1877.

[2] *Vide*, *London Lancet*, 1881, vol. i, p. 149.

[3] "Die Kehlkopfschwindsucht," Leipzig, 1879, S. 69.

The apparent rarity, therefore, of miliary tubercular deposit in the muscular tissue of the larynx induces me to record the microscopical appearances observed in a case which I have recently had the opportunity to examine.

The specimen was taken from the body of a man who had died in an advanced stage of pulmonary phthisis. Cavities were present in both lungs. The epiglottis was the seat of several small ulcerated spots, and there was a large ulcer involving the whole of the left ventricular band, the anterior portion of the corresponding ventricle and vocal cord. The microscopical examination of the ulcer and the portions of the larynx immediately adjoining, presented the usual appearances of tubercular infiltration and ulceration.

In order to determine the changes in the muscular tissue, sections were made through the arytenoid cartilage and the muscles attached to its base. The histological changes consisted (1) in the deposit of distinct miliary tubercles in the substance of and between the muscular fibres, and (2) in alterations in the fibres themselves.

1. In the deeper portions, near the insertion of the muscular fibres into the arytenoid cartilages, were found several large, well-marked, distinctly circumscribed tubercles embedded in the muscular tissue, and wholly unconnected with the tubercular infiltration of the mucous membrane which clothed that portion of the larynx. They lay in the centre of the muscle, between the individual fasciculi, by which they were completely surrounded and which they had forced apart, so that at the situation of the tubercle the muscular bundle presented under the microscope an ampulliform dilatation. The outer fasciculi surrounded the tubercle completely, whilst the central ones passed appar-

ently through its substance, or ended abruptly at its margin in a mass of small round cells. The individual fasciculi were in some places torn completely asunder; in one place, absorbed by the pressure of an outlying tubercle. The tubercles themselves presented the typical appearance of these neoplasms, each containing several giant cells. In one, caseation had produced almost complete destruction of its central portion. In the immediate neighborhood and between the individual fasciculi, in some instances forcing them apart, was an abundant, high-grade round-cell infiltration. The cells were observed, on cross-section, to completely surround the separate fasciculi; at some points they had so invaded the muscular tissue as to leave little of its original structure visible.

2. The most noticeable change in the primitive bundles themselves was an increase in the number and size of the muscle cells. Those bodies were generally enlarged, sometimes irregular in outline, and filled with a granular matter; in others the nuclei were plainly visible. At some situations the muscular tissue showed no well-defined pathological change, beyond, perhaps, a slight increase in number of the nuclei; whilst at others, and especially was this the case in the vicinity of the tubercles, the striation was indistinct, the fibres were broken across, so that the sarcolemma sheath became visible, and in some spots the muscular tissue itself was converted into a granular detritus.

These appearances add little to what has already been observed by Heinze; but as corroborative evidence of the accuracy of his observation, they may prove of some interest to those whose studies have led them into this field of pathological research. As to the import of the granulo-fatty degenerative changes in the muscular fibres, I am inclined to regard them as the result of disturbances of nutrition induced by chronic inflammation, rather than as an integral part of the tubercular process.

A CONTRIBUTION TO THE CARDIO-SPHYGMOGRAPHY OF TRICUSPID LESIONS.

By A. T. KEYT, M.D.,
CINCINNATI, OHIO.

OBVIOUSLY the indications by the graphic method of lesions affecting the right auriculo-ventricular valve must be largely, if not wholly, negative, nevertheless their importance is sufficiently manifest. The value of negative evidence in diagnosis is well understood. Simultaneous cardio-arterial traces in left-side valvular disease give positive indications. Marked aortic stenosis, heavy aortic valves, mitral regurgitation, and aortic regurgitation, cannot exist without distinctive phenomena which express themselves through cardio-sphygmography as diagnostic signs of the respective conditions. In right-side valvular disease such graphic signs are not obtained. Hence, when the question arises as to which side of the heart is the seat of the valvular trouble, the graphic records come in to decide.

Especially it is known how difficult it is by ordinary methods of examination to distinguish between mitral and tricuspid regurgitation. Indeed, it is doubtful whether tricuspid regurgitant lesion has ever been positively recognized during life. Cardiac murmur, so much relied upon in the diagnosis of mitral lesion, is of little aid in the diagnosis of tricuspid lesion; nor are there any physical signs or general symptoms by which the latter can with any degree of certainty be differentiated from the former. The two following cases are in evidence and illustration of the important diagnostic aids cardio-sphygmography may afford toward the recognition of tricuspid lesion.

Case 1.—Mrs. Smith, aged 59 years, came first under my care in March, 1880. She was suffering from dyspnœa, cough, and general dropsy, which were found to be dependent upon organic cardiac disease. There were evidences of considerable enlargement of the heart, and a loud systolic murmur was audible over the cardiac region, plainly emphasized at the left apex, well heard to the left and in the back. The second sound of the heart was clear, and accentuated at the pulmonic site. Heart's action at times irregular; pulse somewhat accelerated, sufficiently ample; no albumen in urine. She continued under my observation without material change of the physical signs, and died, from failure of the circulation, May 8, 1881.

Simultaneous cardio-sphygmograms were obtained May 24, 1880, and February 12, 1881, of which figs. 1 and 2 are examples.

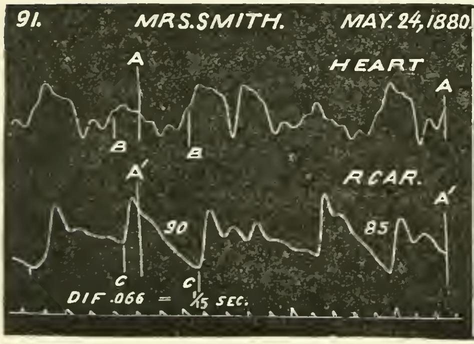

FIG. 1.

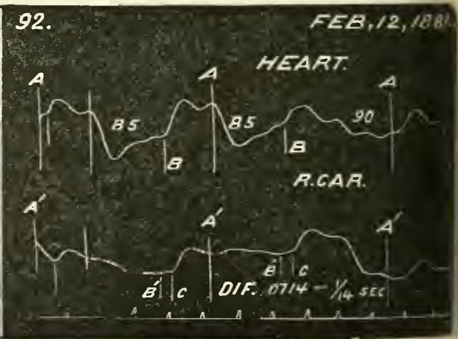

FIG. 2.

The *post mortem*, concerning only the thorax, was conducted by Dr. A. B. Isham in the presence, and with the assistance, of Dr. H. T. Lowry and myself. The pericardium contained a moderate amount of fluid. The heart was generally enlarged. Right cavities and great veins distended with blood, and the former contained firm fibrinous clots. Walls of right ventricle and right auricle greatly hypertrophied—dilated and thickened,—the former measuring one half inch in thickness. Venæ cavæ dilated. Pulmonary valves normal. Tricuspid orifice much dilated and valve incompetent, one of the segments bound down to the sides of the ventricle; the condition of the valve was such as must have permitted free regurgitation during life. Walls of left ventricle thickened to about three quarters of an inch, cavity not dilated. Left auricular walls of normal thickness and cavity of normal dimensions. Mitral valve slightly thickened, but pliable and competent. Aortic valves competent. Texture of heart fatty and friable.

The ordinary signs and symptoms in this case pointed very definitely to mitral regurgitation as the predominant trouble. Especially the auscultatory phenomena declared this dignosis, nor was any thing developed by the usual methods of examination to cast a doubt upon the correctness of this view. It was a case in which the physician, however skilful in cardiac diagnosis, confined to ordinary methods, would inevitably commit the error of assigning the regurgitation to the mitral orifice.

We will now examine the graphic records, and see what light they might have shed upon the case. Allowing for the effects of the respiration and the irregularity shown in the middle of fig. 1, the only positive peculiarity of the cardiac trace is the high auricular wave. The carotid trace is free and ample in fig. 1; shows feebleness in fig. 2. The cardio-carotid interval measures $\frac{1}{15}$ second in 1, and $\frac{1}{14}$ second in 2, with pulse-rate in both 85 to 90 per minute. These intervals are strictly normal.

If free mitral regurgitation had existed, the cardio-carotid interval would have been abnormally long; it would have measured somewhere between an eighth and a quarter of a second. This important fact, established by my own observations, still awaits the case to prove that the lesion may exist without the sign. In the present case I thought I had found the exception to the rule, and stated to Dr. Isham that my sign had failed me, or the case was one of tricuspid instead of mitral regurgitation. I did not feel justified in arraying this simple negative sign against what appeared positive indications of mitral incompetency, and so was almost ready to concede in this instance default of the graphic indication, when the autopsy verified the integrity of the mitral and insufficiency of the tricuspid valve, attesting again the significance and trustworthiness of the graphic testimony.

Case 2.—John W., aged 48 years, a patient in the Cincinnati hospital, suffering from heart disease, and whom I was given the opportunity to observe by the kindness of the attending physicians. He was troubled with cough and dyspnœa, and had a double murmur at the base—the diastolic predominating,—and a systolic murmur at the apex. Apex displaced downward and outward. Simultaneous traces, fig. 3,[1] of the heart and carotid and of the heart and radial were taken February 12, 1881. The

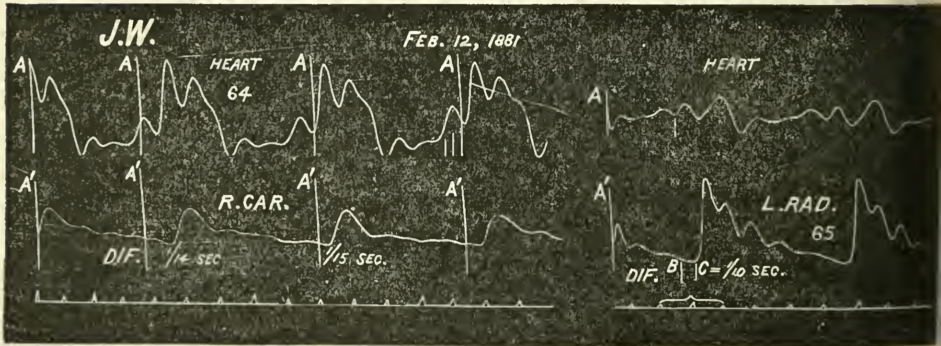

FIG. 3.

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[1] Of the cardiac and carotid traces the former is excellent, while the latter is somewhat undeveloped in consequence of thickness of the tissues overlying the artery. Of the cardiac and radial traces the latter is excellent, while the former is undeveloped by a faulty application of the base. It is to be regretted that the last imperfection had not been corrected by a repetition of the experiment. However, the traces all show distinctly, and in proper position, the basal points, and so permit of correct determinations of the time-differences.

man died, and a *post mortem* was made April 19, 1881. From the hospital notes and personal inspection of the specimen, I gathered the following history : Marked dilatation of both ventricles and hypertrophy of ventricular walls. Right auricle dilated, and walls thickened. Tricuspid orifice contracted so as to admit only the tip of the little finger ; valve thickened, and manifestly insufficient, as well as directly obstructive, during life. Pulmonary valves normal. Mitral valve normal. Aortic valves slightly thickened, and one contained a small calcareous plate; yet they preserved their normal form, and were evidently competent during life. Aorta dilated from near its origin to its bifurcation into the iliacs ; especially the transverse and descending arch was dilated into a cylindrical pouch of considerable size ; aortic walls thickened, rigid, and pervaded with calcareous plates varying from a quarter of an inch to an inch in diameter.

In this case no definite diagnosis had been arrived at ; especially the tricuspid lesion had not suggested itself. The double murmur at the base was thought to indicate aortic obstructive and regurgitant lesion ; and the systolic apex murmur, mitral regurgitation ; cardiac hypertrophy only was distinctly recognized. A correct appreciation of the graphic showings would have demonstrated the absence of grave left-side valvular lesions. If aortic stenosis had been present the pulse-trace would have been characteristically deformed, and not free and shapely, as shown, notably in the radial. If there had been obstruction at the aortic valves, or regurgitation at the mitral valve, one or both, the succession of the arterial pulses on the heart would have signalled abnormal delay instead of the intervals shown. If free aortic insufficiency had existed, the carotid and radial pulses, besides presenting the characteristic form of this lesion, would have appeared much sooner after the systole of the ventricle than the time indicated. And further, if pure mitral contraction[1] had existed to produce such distress, ventricular systole would not have shown such development, nor the arterial pulse such fulness and amplitude.

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[1] Opportunity for tracing mitral contraction has not offered, but recent experiments on the schema indicate that the pulse is abnormally delayed, also, in this condition.

Then, as the evidences of grave valvular disease were unmistakable, and the graphic records proved the integrity of the aortic and mitral, the localization was driven to the right-side valves, and attention to the signs of the graphic method would have secured a diagnosis which could not have been arrived at by other means.

The traces sufficiently indicate hypertrophy of the left ventricle, but they demonstrate also the presence of auricular hypertrophy. The latter information is furnished only by the graphic method; when either auricle is hypertrophied there will be in the cardiac trace a correspondingly high auricular wave.

Finally, we find in the traces evidences bearing upon the arterial rigidity. The cardio-carotid interval being $\frac{1}{15}$ second, and the cardio-radial $\frac{1}{10}$ second, would give only $\frac{1}{30}$ second for the carotid-radial interval. This is less than half the average normal time-difference between these points in healthy adults, and as arterial rigidity is known to be the greatest of all factors increasing pulse-wave velocity, such unusual speed as is here signalled indicated that the arteries of the upper extremity partook of the rigidity so strikingly displayed in the aorta; and this rigidity extending in the innominate and carotid was the undoubted cause of the unduly short cardio-carotid interval. It is probable the pulse-wave velocity will come to be accepted as a test and gauge of the quality of the arterial walls as to their degree of rigidity or pliancy.

The diagnostic signs furnished by cardio-sphygmography go hand in hand with the evidences of auscultation and percussion, correcting, extending, and completing the latter, and give to cardiac diagnosis unparalleled precision and perfection.

THE PATHOLOGY AND PHILOSOPHY OF SEA-SICKNESS.

BY C. PITFIELD MITCHELL, M.R.C.S.

DURING the summer months of last year, as surgeon on the Atlantic mail service, I was afforded opportunities for studying the very interesting chain of pathological events exhibited in sea-sickness, and sharing the existing want of a rational synthesis of these phenomena, I have colligated the facts and worked out the conclusions which I propose to embody in the present paper.

An investigation of the pathology of this disease is facilitated by the absence of circumstances which usually veil the relations of cause and consequence. Unlike many kindred inquiries the conditions are simple. In the first place, the oscillating of the vessel on its transverse and longitudinal axis, as the primary initiator of the functional disturbances, stands out with unmistakable reality; then this oscillation is roughly measurable in its increments and decrements, and varies together with measurable increments and decrements in the pathological effects. In other words, the factors and their products may be known quantitatively, which is seldom possible in the investigation of morbid phenomena outside the laboratory. The effects, too, are few in any given case, and but little complicated in their mutual connections, and may also be observed in an indefinite number of instances under similar conditions.

Although my own observations will form the groundwork of the following reasoning, yet since these are not fundamentally at variance with those of others, the statements of fact may conveniently be more dogmatic than would be otherwise permissible.

Between the symptoms first felt in the well-marked, and those present alone and throughout in the mildest examples, there is a likeness in kind; in both they are subjectively cerebral. Disturbances of the intellectual centres are known by confusion of thought, mental torpor, an inability to fix the attention, and volitional weakness; changes from the usual currents of the sensations and emotions are evidenced by frontal and occipital headache, cerebral throbbings, intolerance of sensory impressions, irascibility, dejection, and despair. The physiological susceptibility existing, there will be faintness and vertigo, nausea and vomiting, and an inclination to remain recumbent and to sleep. A physical examination would then elicit the following conditions: pallor of the face, coldness and clamminess of the extremities, the pupils probably normal, the circulatory and respiratory functions depressed, the pulsations being slow, frequent (?), and small, the arterial tension low, the temperature normal or subnormal, the tongue clean or covered with a thin whitish fur, thirst, the bowels constipated, urine scanty and concentrated. Such, abstracted from my own experience, is an average of the signs to be met with in all stages of the disease. It will be necessary to dwell upon some of these signs in detail to point out some variations from the average, the frequent co-existence of other independent and obscuring symptoms, and the circumstances under which they severally arise. The subjective phenomena vary, of course, slightly in character, and widely in degree, with the individual organism and the degree of pitching and rolling. Those especially predisposed may be quite unable to remain in the upright posture. I can recall four cases where the fainting amounted to asthenic syncope. In these cases there appeared to be a deficiency from the beginning in the reactive forces of the body, and the sickness and want of food were unusually protracted.

Generally the vomited matter consists of watery fluid, with more or less bile intermixed. Exceptionally, if the straining be excessive, slight hæmatemesis may supervene. The circumstances which influence the degree of vomiting may be mentioned incidentally, that the argument to follow may not be anticipated. In the first stage the epithelium of the tongue undergoes little change ; later the organ is thinly furred, and the tip speckled. Gastric catarrh, so frequently a concomitant of sea-sickness, is probably determined by exposure to other unhealthy conditions of ship-life, chief among these being impure air and water, and stale food. That it is not a primary symptom, and may have an independent origin, can be known and inferred from the facts that it usually appears late in the attack, *i. e.*, after exposure to the above conditions, is often persistent during convalescence, and is found among those who enjoy an immunity from sea-sickness. Salivation is present in a considerable percentage of cases, but whether it properly belongs to the group of symptoms directly resulting from the ship's motions, is, I think, uncertain. The best examples I have seen were associated with gastric catarrh.

Of twenty-nine recorded cases—a fraction only of the number actually observed—constipation was present in twenty-eight, in the remaining one early diarrhœa and subsequent constipation. So constantly are the bowels confined from the commencement and during the continuance of the attack, that when they are not so it may always be confidently attributed to extraneous causes. And this, too, after duly allowing for the presence of the hydrated peroxide of iron in the water used for drinking and cooking. The water for such purposes is stored in iron tanks, which, unless frequently cleaned out, contain a thick sediment of iron rust, very easily stirred up in rough weather. That used for drinking is sometimes filtered, but not so the water used for cooking. This, as one of the causes of costiveness at sea, especially in those who are not sea-sick, does not appear hitherto to have been noticed. The fæces may show the characteristic staining. The average temperature (buccal) drawn from the notes of twenty-five cases was 96°.7; the maximum being 99°.2 and the minimum 94°.6. Respecting the differences of temperature, the general conclusions may safely be made, that an elevation is either indirectly caused by the co-existing constipation or has some origin unconnected with the cause of sea-sickness, and that if the sickness be moderate and quickly rallied from, the temperature will be normal; if, on the other hand, severe and lasting, it will fall. There is no sign more distinctive than the enfeeblement—it appears to be nothing more—of the circulatory and respiratory functions. Occasionally, where vomiting is absent and some other disease is suspected, the finger on the pulse may suffice to decide the diagnosis. The average pulse-beat in twenty-eight cases was found to be 78; the highest being 116, and the lowest 56. This, as a typical average, should be accepted with reservation, for the cases recorded were all among the emigrant class of passengers, whose hygienic and social surroundings were likely to complicate the pulse-beat of sea-sickness. Also, the observations were necessarily taken indiscriminately, with the patients lying, sitting, or standing. It is important to note the invariably low tension of the pulse. The urine is diminished in quantity, of high specific gravity from a relative increase of normal solid constituents, without albumen or sugar.

The next step is to bring into view the series of changes by which the external incident force works that internal pathological state of which the symptoms set forth are the outward and visible signs. The veritable *questio vexata* of sea-sickness must now be examined, and the right answer discovered, before a complete and scientific *rationale* of the disease can be attained. A brief retrospect of past inquiry in this direction will afford light and guidance in the present search.

Sir James Alderson[1] cites an experiment made by Dr. Wollaston, 60 years ago, to the effect that "if a barometer be carried out to sea in a calm, the mercury will rest at the same height as when on shore; but when the ship falls by the subsidence of the waves, the mercury is seen apparently to rise in the tube which contains it." Using this experiment to explain the pathogenesis of sea-sickness, he remarks that "the approximately rigid brain and vessels are carried downward, the blood remains by its own inertia, and the consequence is to crowd blood into the vessels of the brain, and so press with increased force, producing a certain shock; this shock and the attendant pressure produce sickness and vomiting." Dr. Pollard,[2] pointing out the fallacy involved therein, rightly observes that "at sea the falling of the ship is due to the action of gravity. There would be no tendency, therefore, for the mercury in the barometer, or the blood in the body, to commence falling later than the tubes which contain them. They would all begin to move at the same instant. It is, nevertheless, a fact that the action of the waves does cause the mercury in the ordinary barometer to oscillate when the sea is rough. As already shown the mere falling and rising of the ship would not account for this; it is the sudden striking of the ship by the waves which in rough weather jerks the mercury up the tube; and in order to reduce this to a minimum, certain ingenious modifications have been introduced in the construction of marine barometers." Dr. Pollard, therefore, rejects this explanation; he thinks "the most probable theory of sea-sickness is that held by Dr. Carpenter, Mr. Bain, and other writers, who consider that the mental and bodily prostration and the other symptoms arise from continued action on the brain of a certain set of sensations, more particularly the sensation of want of support. The phenomena of sea-sickness appear to be due to the constant repetition of this feeling of loss of support consequent on the pitching and rolling of the ship, more particularly the former." Without diverging to show the incompatibility with the admitted facts of this hypothesis, and the cognate one of "association of sensations," it will be more profitable to proceed to find, if possible, a mechanical interpretation upon which may be constructed a synthesis of all the data we possess relating to sea-sickness.

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[1] "Observations on Sea-Sickness." *British Medical Journal*, March, 1872.

[2] "Remarks on the Pathology and Treatment of Sea-Sickness." *British Medical Journal*, June 8, 1872.

First should be considered, in juxtaposition, the nature of the force in action and the body acted upon. The rise and fall of the vessel in pitching and rolling is seen to be a rhythmical, *molar* motion, having relatively large amplitudes of moderate uniformity. Now whatever be the degree of motion communicated to the organism as a whole, and to its parts in respect of one another, such communicated motion must partake of this character; it must *in the first instance* also be molar. Molecular changes in the brain propagated through the organs of sight may be excluded, for sea-sickness occurs whether the eyes are opened or closed. The body being composed of fluids and solids, of parts having different mobilities, we shall look for disarrangements among these as the primary internal changes induced by movements of the whole through space. *Molecu-*

lar disarrangements, if induced at all, must be secondary. But the organs and viscera are intimately joined together by their correlated structures, and by connective tissue, and much of the blood circulates transversely to the direction of the incident force, therefore only that moving vertically will undergo any appreciable displacement.

For our present purpose we may superficially regard the vascular system as consisting of arteries and veins ascending from the heart to the head and neck, and arteries and veins descending from the heart to the lower extremities. In both cases the vessels leave the heart as tubes having large sectional areas, and terminate at the peripheries as tubes having small sectional areas. How then will the blood circulating in these vessels comport itself when the body is subjected to rhythmical elevations and depressions? As the body falls, the blood-pressure both of descending and ascending vessels will remain unchanged, for vessels and contents will gravitate together, and as the body rises, to the descending set of vessels and contents the motion will be transmitted equally throughout the system—arterial, capillary, and venous,—and the balance will not be disturbed. What now will be the effect upon the blood of the superior vessels when the ship ascends? In so far that the arterial trunks are dependent, and end above as capillaries, and the walls are rigid relatively to the contained blood, we should anticipate a recession of blood to the aorta; but from the end of one systole to the commencement of the next the semilunar valves are closed, and the supported column of blood would receive an upward acceleration, together with the heart and vessels, and during systole, the heart rising with the body will impart this motion to the blood at that moment being propelled; therefore, the tension throughout the arteries will probably not be directly influenced. Note, however, the effect upon the venous blood-pressure. When the body rises, the descending blood will be relatively accelerated, and the pressure will subside at the periphery. That this will happen seems inevitable ; the upward motion of the body and vessels cannot be at once communicated to the enclosed blood, for the veins increase in calibre toward the heart, are without valves, and are not normally distended to their full capacity. In order to understand the mechanism more fully, attention should be given to the researches of Dr. Moxon, "On the influence of the circulation upon the nervous system."

The following passage[1] will serve to make clear the point we are considering: "The way by which the cerebro-spinal fluid enters the cranium and brain is by a relatively wide and free passage; while the way by which the blood enters the cranium and brain is by the relatively narrow passages of the capillary arteries ; and if the circulation be feeble, the heart may drive the blood less forcibly to the texture of the brain than the venous pressure in the spinal canal drives the cerebro-spinal fluid into its cavities, so that when the head is raised and the venous blood falls away more readily from within the skull, the cerebro-spinal fluid may outstrip the arterial blood in the race to supply the place. And thus the ventricles of the brain may tend to fill with water more quickly than the substance of it with blood, and so the brain blanches and the person feels giddy." Knowing, then, the effects upon the cerebral circulation of a single upward movement of the body, we are in a position to follow out the sequences, under certain physiological conditions, when the motion is compounded of elevations alternating with depressions. At the close of the first ascent the blood in the arterial capillaries is lagging behind that in the venous capillaries ; and during the descent, by increased cardiac action equilibrium is partly restored. With the next ascent the pressure in the venous capillaries is again lowered, but now more readily than before, for the heart, assumed to be incompetent to maintain the balance from the beginning, has been further incapacitated by the previous drain upon the cerebral vessels, and by the interference with its normal rhythm in the medulla oblongata, and probably by varying pressures in the right auricle[1] and sinus. The series of motions continuing, it is easily seen that a relative downward impetus to the venous blood and an increasingly uncompensating rise in the arterial blood must end eventually in anæmia of all parts of the brain. And this completely harmonizes with the mode of onset of sea-sickness ; it is not sudden, but gradually progressive. It thus appears that the cause of sea-sickness is in the *rhythm* of the motion to which the physiological subject is exposed. If the upward force were continuously in action the initial disturbance would soon be readjusted through the heart, which in this case would not be distressed by interferences with its organic rhythms.

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[1] Croonian Lectures : *British Medical Journal*, April, 1881.

Taking with us the above deduction, and following out its corollaries, we may compare these side by side with the inductions.

A sufficient circulation of healthy blood in the cerebrum being one of the conditions essential to normal psychical processes, viewed only as to their quantity, if the circulation fall to an undue level the strength of the intellectual actions will also decline. As we have already seen, in sea-sickness the thoughts are sluggish, the attention cannot be focused, the predominant wish is not to do any thing, and there is a disposition to sleep.

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[1] This inference is interestingly corroborated by Dr. Gaskell's results of his novel experiments on the action of the frog's heart, brought forward at the recent International Medical Congress, and upon which I have fallen since these lines were written. He remarked that "any influence acting upon the auricles and sinuses alone which can alter the rhythm, such as heat, cold, atropin, muscarin, etc., makes the rate of both auricles and ventricles synchronously quicker or slower, while the same influences affecting the ventricle alone produce no alteration in the rate of rhythm of either auricle or ventricle. We may conclude, then, that *the cardiac muscle in the normal heart contracts to each impulse which reaches it from the sinus or auricles*, so long as the muscular tissue is in a condition in which it is ready to contract."

The accompanying emotional states also become intelligible by joining this physiological principle with a certain law of mental evolution. The nervous plexuses by which the conduct is adjusted to social requirements are among the last evolved in the race, are among the least permeable channels for the discharge of nervous energy, and, when the pressure is high, are the courses taken by the overflowing force; if, then, the pressure fall, if much of the circulatory energy—the expression is used advisedly—be drawn off with the blood, the residue will run through the older and better organized channels, the plexuses for aggression and defence. In like manner, depression—note the word—of spirits is interpreted.[1] The facts of sea-sickness in this connection may be recalled. Indications of ill-temper I have noticed so frequently in both sexes, and in those distinguished at other times for their amiability, that I have learned to look upon such manifestations as pathognomonic signs, while the dispiriting influence of sea-sickness is proverbial.

As coöperating indirectly to produce these subjective states, the molecular vibrations set up throughout the body by the action of the propelling apparatus of the vessel, and the muscular tensions caused by the latter's movements, as a whole, should not be overlooked. Among the effects of these forces which may be said to do work upon the organism while this is passive, are doubtless the indolence and large appetites of those who are in health at sea. Although paleness of the face is possible apart from cerebral anæmia, yet with the inference that all the structures above the trunk are depleted, the fact agrees. The functional strength of the heart and lungs—and not unlikely of the vaso-motor nerves,—being derived from the medulla oblongata and ganglionic systems, will wane if the blood circulating in the medulla be much diminished. This, which physiology teaches, pathology abundantly ratifies. And to the extent that vomiting is admitted to be a symptom of cerebral anæmia, this also might be taken as verifying the deduction. If, however, the relations of the pneumogastric and phrenic nerves to the large vessels in the thorax be borne in mind, we shall perhaps be led to think that these phenomena are caused ento-peripherally. The right pneumogastric nerve passes between the right subclavian artery and vein ; the left, between the left carotid and subclavian arteries and behind the left innominate vein ; and the phrenic nerves pass over the first part of the subclavian arteries, between them and the subclavian veins. Now upon some of these structures the incident force will be concentrated. As the blood-pressure at the venous periphery falls, the pressure at the centre will rise, the heart being unable to distribute the blood of the surcharged venous trunks ; and though the facts show a corresponding fall in the arterial tension, it is at least possible that the normal relations of these nerves to these vessels may be so changed that the functions over which the nerves preside may be perverted in the manner observed. The distribution in the thorax of the little-known sympathetic plexuses only helps at present to confound our knowledge of the actual factors. The intensity of the vomiting in sea-sickness points with some cogency to an ento-peripheral genesis of this symptom ; it is difficult to reconcile with an anæmic state of the brain, so large an evolution of nervous energy from the central system as the vomiting in this disease implies. Continuing to follow out the sequences, as the arterial tension throughout the body slackens, and the general circulation flags, conspicuous functional changes may be sought at the external and internal peripheries. And there in sea-sickness they are found. The hands cold, clammy, and bluish ; the bowels deficient in natural secretions and peristaltic power ; the eliminations of the kidneys less in quantity and of greater density than normal, are clearly among the immediate consequences of an imperfect circulation. But here again the antecedents are compound. Since the performance of every organic function is aided by the general diffusion through the system of continuous centrifugal discharges of nervous energy (Spencer), a considerable subtraction from the chief source of these, the brain, will be a derivative and conjoint cause of this general functional quiescence.

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[1] See Spencer's "Principles of Psychology," vol. i, pp. 603 to 605.

The observed low temperatures are similarly explicable. There cannot be ascribed to thirst in sea-sickness the meaning we give to it in most other diseases ; general tissue-changes and aqueous loss by the lungs, skin, and kidneys being reduced, this feeling must be otherwise created. Sensations of this class probably depend upon the physical state of certain sensory end-organs of the alimentary canal which have immediate contact with alimentary material, so that if, as by constant vomiting, the fluids normally bathing the mucous membrane be exhausted, thirst may be produced irrespective of the general needs of the organism. Illustrations of this are found in the temporary relief to thirst afforded by simply moistening the mucous membrane of the mouth, and in the thirst, out of all proportion to the quantity of fluid lost, in the smoker who expectorates. In the present instance the sensation may certify to a real want, for excessive vomiting may preclude even the ingestion of such water as is needful for the most vegetative functions.

Sundry facts, purposely left for consideration until this stage, may now be presented as additional support to the foregoing argument, and at the same time receive their own elucidation. In all cases of sea-sickness, probably without exception, a change of posture from the perpendicular to the horizontal is very shortly followed either by a total cessation of all the symptoms or some measure of amelioration. With many persons who are fairly comfortable while reclining, even a slight elevation of the head and trunk suffices to provoke a return of the malady. That relief should ensue on lying down is so obviously in harmony with the views advanced, that to mention it is enough. Still the fact remains that in some individuals partial sickness continues notwithstanding. This may appear *prima facie* to be somewhat contradictory, but by a little reflection it may be seen that when the oscillations are extreme—and it is then that the exceptions occur,—if the horizontal position be assumed the conditions favoring sea-sickness will not have been wholly removed. And when the perturbations of function are profound, according to the interpretation here given of their pathogenesis, the time occupied in recovering, when the perturbing force is no longer present, may be expected to vary with individuals. Instances of severe relapse clearly traceable to the taking of saline and other purgatives came under my notice from time to time. In my own person I suffered twice in this way, and sought the explanation in the increased cerebral anæmia and cardiac debility which must follow an influx of blood to the intestinal canal.

What are the physiological processes involved in recovery from sea-sickness? Here we are in the region of conjecture, and at present cannot hope for more than a partial solution. Taking recovery or the natural restoration of functional equilibrium as one manifestation of the law of adaptation formulated by Herbert Spencer (" Principles of Biology "), we shall be able to comprehend the dynamical processes in a general, if not in a special, sense. Plainly, the case before us is one of *direct* adaptation, and an example of Mr. Spencer's second form of organic equilibration. By this we know that "any disturbing force, working an excess of change in some direction, is gradually diminished and finally neutralized by antagonistic forces, which thereupon work a compensating change in the opposite direction, and so, after more or less oscillation, restore the medium condition" ("First Principles"). With this general and scientific conception of the *vis medicatrix naturæ* in sea-sickness, a more special one comes within reach. Adequate *a posteriori* reasons will presently be adduced for believing that cardiac weakness is the essential physiological fault; the heart is unable to cope with a new force whose tendency is to overthrow the equilibrium of the cerebral circulation. There is an excess in the incident force which cannot be counterbalanced. We may learn something by inquiring what becomes of this excess? It is dissipated in working the characteristic functional changes in the special ways already described; the heart's contractions are restrained, the sensible and insensible motions of the intestines are arrested, the evolution of nervous energy in the cerebrum and the molecular motions throughout the body are reduced to a minimum, and the whole organism, in respect of its voluntary movements, is brought to rest. If this fails to tell us *how* recovery is sooner or later compassed, it at least answers the question *why*. The heart and brain are finally enabled to equilibrate themselves to the force whose rhythm has overthrown them, because the brunt of this force is shared by all the other organs of the body. If it were possible for the surplus force to remain undistributed while the organs upon which it falls are unable to poise it, recovery could not take place, the heart must stop, the equilibration would be death. That the ultimate adaptation consists in an integration of energy at the places of incidence of the external force, is to be inferred from the general principles of equilibration. The strength and permanency of the adaptation will be expressed by the product of the incident force, and the time during which it is in operation. In my own case, on the first voyage, after an illness lasting three days, adaptation was complete, but not sufficiently organized to continue throughout a week's stay on shore, as proved by some sickness at the commencement of the second voyage, and again, still less severe, after a like interval on the third voyage. Given a constitutional predisposition to sea-sickness, however deeply ingrained be the acquired inurement, according to the doctrine of evolution, if the modifying force be removed, the *status quo* will gradually be approached, though never reached.

Other things being equal, sea-sickness is moderate or severe relatively to the amplitude, period, and synchronism of the ship's oscillations. The "pitching" motion and the motion of a small boat in a "choppy" sea are particularly searching.

The nervous reco-ordinations assumed to have taken place in the newly adapted organism will be an adjustment to rhythmical motions not widely deviating from a mean; hence an adaptation to the swelling waves of the Pacific Ocean may fail on the short waves of the English Channel, as sometimes happens. Since acquired modifications of structure and function are transmitted to offspring, the children of those who pass their lives at sea may inherit the adapted nervous systems of the parents.

What functional or structural peculiarity protects the infant from sea-sickness? Not unlikely the relatively unadvanced development at this age of the brain and its blood-vessels. Probably in infancy the integration of the visceral functions in the cerebrum is still incomplete.

The answer it has been found necessary to assume to the question: Why are some people sea-sick and others not? may now be made good. Already some grounds have been given for thinking that functional differences underlie differences of susceptibility to sea-sickness. Then it may be prevised that where the functional momenta and the potential forces of the body are below par, the moving equilibrium will be relatively unstable and easily disturbed. This we shall find inductively substantiated. Boys and girls at the age of puberty, when the expenditure for daily activities is largely supplemented by that for growth and differentiation, have appeared to me to suffer especially; females are more prone than males, and we know their physiological reserve to be generally less; temporary weakness and debility seem frequently to determine an attack; and so-called "nervous" people, with weak vascular systems, rarely escape. Contrarily, when the functional momenta and the potential forces are at par, the moving equilibrium will be more stable and not easily disturbed, which ought to be exemplified in those who are *not* liable. And so it is. Allowing for qualifications, men and women at adult age, whose health and physique are high, suffer the least frequently and the least severely; and, generally, it may be said, the better the animal, the more complete the immunity. (See Dr. Beard on "Sea-Sickness and its Treatment.") From this and what has gone before, we shall be justified in concluding that sea-sickness depends on the functional stability of the heart and brain; depends on the ability of these organs to immediately resist the antagonistic force which falls upon them. This conception of sea-sickness is submitted as the basis for a scientific treatment.

A CONTRIBUTION TO THE STUDY OF ANÆMIA.

By FREDERICK P. HENRY, M. D.,

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THE value of the hæmacytometer as a means of diagnosis, depends upon the wonderful recuperative power of the blood as regards its proportion of water. But for this tendency toward the maintenance of a certain quantity of blood in the vessels, irrespective of quality, the instrument would be of little value beyond determining the proportion of white cells to red ; and where this recuperative power is interfered with, the examination, unless made by one who has had some experience in such matters, might be in the highest degree misleading. It is interfered with in choleraic conditions, that is, in states of the system in which the waste of water is greater than the supply ; and also in conditions of imperfect ingestion and absorption of water, the waste of this fluid remaining at or below the normal.

A marked example of the latter of the two conditions above referred to, was furnished by a case of stenosis of the cardiac orifice of the stomach, which came under my notice at the Episcopal Hospital, in April, 1881.

William G., a Scotchman, æt. 56, had first experienced difficulty in deglutition five months before admission. He was then an inmate of a soldiers' home in Virginia, and the symptoms first appeared immediately after excessive vomiting casued by a debauch. His family history was good. He had never had syphilis. He was greatly emaciated, having lost fifty pounds in five months, and suffered greatly from excessive thirst. He declared that water would not pass the stricture. With much difficulty a drop of blood was squeezed from a deep needle-puncture of the finger. It was very dark in color, and undoubtedly inspissated. An enumeration gave the following result :

No. red cells per cubic mm., 5,525,000.

Proportion of white cells to red, 1 to 550.

The normal average of red cells per cubic mm. being about 5,000,000, the above figures would have indicated an excellent state of blood nutrition, had it not been evident, from the clinical history and the physical state of the blood, that its proportion of water was greatly diminished. The term oligæmia would properly describe the condition then existing.

The patient was nourished with enemata until my colleague, Dr. Forbes, succeeded in passing a bougie into the stomach, after which he was able to take liquid food by the mouth, and rapidly gained flesh and strength. He soon obtained permission to go out on a pass, but returned exceedingly drunk, and was discharged.

As I did not make another count in this case, I can only surmise that the absorption of water and improvement in the patient's general condition were coincident with the establishment of a high degree of anæmia (oligocythæmia), a state of affairs that might be appropriately styled a medical paradox !

The more accurate diagnosis of anæmia attained through the use of the hæmacytometer, has led to certain modifications of our views regarding the pathology of several affections. By the term pathology as here used, I intend to imply the relation between the clinical history of a disease and its anatomical lesions real or supposed. By the process of blood-cell counting I have in more than one instance demonstrated that well-marked neurasthenia may exist independently of anæmia. I have also been able to confirm the observation that symptoms formerly attributed to plethora are often due to anæmia. As to the existence of plethora I am extremely sceptical. I have never seen any morbid condition whatever that could be ascribed solely to an excessive number of red cells in the blood. I recognize a condition common to hearty eaters of animal food, which has been called plethora, but attribute its symptoms, for the most part, to albuminoid indigestion, and would classify it under the head of lithæmia. This condition has been admirably described by Fothergill.

The following case illustrates what might be termed spurious plethora.

Miss D., æt. 19, came to me complaining of a daily flushing of the face, coming on after meals, most marked in the evening, not accompanied with headache, and followed by pallor and slight sense of chilliness. At other times her extremities are warm; hands usually moist. The flushing was so annoying that she abstained from going out in the evening. On one occasion, a year before I saw her, an attack came on while she was in church—an evening service,—and was followed by a severe attack of syncope. A physician was sent for who did not think it safe to take her home until midnight.

The girl, when I saw her, was the picture of health, her complexion being remarkably good, her eyes bright, and her appearance in every respect that of a well-nourished individual. In spite of a large appetite she had a constant sense of weakness. Her menses were perfectly regular. As she had lived in a malarious district and there seemed to be a certain periodicity in the attacks, I prescribed a pill of quinine and valerianate of zinc.

A week later she returned and reported only a slight improvement. I then counted the blood globules with the following result.

No. red cells per cubic mm., 3,995,000.

Proportion of white cells to red, 1 to 530.

This showed the case to be one of decided anæmia, and I prescribed wine of iron and arsenic. Three weeks later I found her wonderfully improved. The flushing of the face had ceased entirely; the sense of languor had left her, and her spirits were any thing but low. I am confident that such cases have often been described and treated as plethora. At the same time I admit, that some physicians of large experience have learned empirically that they are benefited by hæmatinics.

A desideratum in the diagnosis of anæmia is an instrument that will determine the specific gravity of small quantities (a drop or two) of blood. In the first of the two cases reported above, there was no difficulty in deciding that the specific gravity was abnormally great, but in slighter degrees of similar conditions this could not be determined. A color test is not corrective of the unavoidable inaccuracies of an enumeration, and is alone not nearly so accurate, as I have elsewhere argued.[1]

In conclusion, I would refer to an interesting observation made by myself for the first time, so far as I know, upon a property possessed by the red blood cells of permeating porous substances. I take the opportunity of introducing it here, although it has no direct bearing upon the subject of anæmia.

On Sept. 20, 1881, I performed the following experiment: After counting the blood cells in the usual manner, I threw upon a filter the same mixture from which the count was made, and then counted the cells of the filtrate. The first count gave the following result: No. red cells per cubic mm., 6,250,000; the second, that of the filtrate: No. red cells per cubic mm., 1,770,000.

The first count is about three and a half times greater than the second, and, therefore, more than one third of the red cells passed through the paper. There is little doubt that if larger quantities were dealt with, a still greater proportion of cells would pass. The whole amount of fluid thrown upon the filter in my experiment was 2,000 cubic mm. (about half a drachm), and although the utmost care was taken to place the liquid exactly in the centre of the cone formed by the folded filter paper,[1] the immediate effect of the operation was the spreading of the fluid by imbibition on all sides upward for the space of an inch or more. This was equal to a reduction of the fluid to about one half of its bulk. If instead of half a drachm, I had been dealing with several ounces, I have no doubt that a much larger proportion of cells would have passed through. It is scarcely necessary to say that in making such an experiment the blood must first be mixed with a substance that will prevent its coagulation. I made use of the fluid which I am accustomed to employ in my blood-cell counts, a solution of sulpho-carbolate of sodium, sp. gr., 1020.

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[1] Cartwright Prize Essay, 1881.

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[1] The paper used was the imported German filter paper.

FURTHER NOTES ON A CASE OF FILARIA SANGUINIS HOMINIS.

BY CHARLES E. HACKLEY, M. D.,

NEW YORK.

FROM February to July, 1882, there was in the N. Y. Hospital, D., who is "Case 1," described by Dr. Robert Abbe in the *N. Y. Med. Journal*, Feb., 1880. The change in his case has been so slight, and the increase of accurate knowledge of the cause of his disease is so scanty, that perhaps there is little reason for reporting it again.

But at present the inclination to refer many diseases to germs, ferments, etc., in the system, may induce readers to be lenient in their criticism of our effort to maintain an interest in a case presenting such a decided organism in the blood.

The case is that of a man 37 years old, born in the West Indies, raised there, and living there, except at intervals, until within 7 years. During his life he has had several attacks of chyluria, passing from the normal amount to 100 oz. of urine in 24 hours. These attacks would pass off almost as suddenly as they came, and the patient would be moderately well. He has been subject to abscesses, and when he entered the N. Y. Hospital he was covered with boils; but where these were emptied we could discover no worms in their contents; nor does he know of any worms having ever been discharged, or that the occurrence of his abscesses has borne any relation to the subsidence of his chyluria.

Perhaps it may not be amiss to give a synopsis of the present views of the cause of chyluria, and of some diseases supposed to have the same etiology, such as elephantiasis, lymph-scrotum, some cases of leprosy, hæmaturia, phlebitis, furuncles, and hydrocele.

In 1872, Dr. T. R. Lewis found small worms in the human blood. They were about $\frac{1}{75}$ of an inch long, and $\frac{1}{3500}$ in diameter. From one to thirty were found in a drop. This worm he named filaria sanguinis; it had previously been discovered in the urine.

In the stomachs of mosquitoes, which had fed on a patient with filaria, Dr. Manson found from ten to one hundred and twenty specimens. He asserts that the mosquito (or at least *a certain* mosquito) does not digest *human* filariæ, but will digest those from dogs, which come from a different worm. Dr. Manson says: "The female mosquito, after gorging itself with human blood, repairs to stagnant water for the purpose of digesting the blood, and depositing its eggs. During this period, which lasts four or five days, the filariæ undergo remarkable changes. Subsequently, in a more perfect state they escape into the water, and in this advanced stage they are conveyed into the human blood along with the water as drink."

Lewis says: "When the insect is caught, shortly after feeding, and the contents of its stomach examined microscopically, the hæmatozoa, if present, will be observed to manifest very active movements, which may possibly continue several hours on the slide."

These movements are like those of the common earthworm, when the angler is trying to put it on a fish-hook; and the filaria looks something like an angle-worm, but under a high power it shows a delicate filament or membrane projecting from each end (as represented in the accompanying wood-cuts).

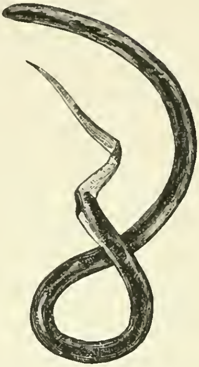

FIG. 1.—From a photograph of filaria from Dr. Mackenzie's patient, taken by Prof. Wm. T. Belfield. Filaria dead and stained. The so-called caudal sheath is well shown.

The filaria having progressed somewhat from its state in man's blood till it enters with water into his stomach, about its development after that, nothing is known. But concerning the parent, Dr. Bancroft, in 1877, says: "The worm is about the thickness of a human hair, and is from three to four inches long. By two loops from the centre of its body it emits the filariæ, described by Carter, in immense numbers." Bancroft removed a worm from a lymph abscess of the arm, and four from a hydrocele. It is supposed that the mature worm is located somewhere in the lymph system, and that the filariæ found in the blood are embryos thrown off in immense numbers from this parent.

In our patient, as in many other reported cases, the filariæ could only be found in blood drawn at night; Dr. G. L. Peabody, who frequently examined him, says he has always failed to find them in blood drawn by day, or in any portion of the urine.

It is said that if the patient is kept up at night and sleeps by day, the habits of the filariæ are also reversed. Attempts have been made to explain this intermittent appearance by saying: (1) that the parent throws off a new brood every

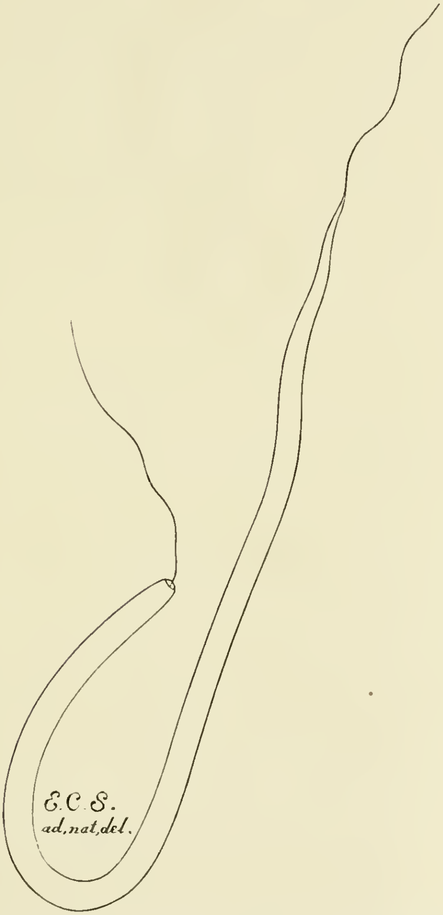

FIG. 2.—Filaria from Dr. Hackley's patient, as observed on April 26, 1882. Drawn and measured under a No. 7 Hartnack objective, magnifying 500 diameters. Actual length of worm, exclusive of filaments, about .03 mm. ($\frac{1}{75}$ inch). (The cephalic filament is drawn too long, and does not taper sufficiently.)

24 hours; or (2) that the filariæ collect in the lymphatics or some internal organ during the period they are absent from the blood. Both theories are open to question.

Some observers in the East say nothing about this daily disappearance, so perhaps it is not universal. It is said that embryos withdrawn from the blood in the evening did not disappear for 170 hours, while those taken in the morning did so in 108 hours.

If these filariæ are embryos and do not propagate, but come either in immense broods or by constant streams from a parent worm, and this parent only enters the system by drinking water containing advanced filariæ in hot countries, we must acknowledge that the parent may be a long-lived animal. Our patient has had his present attack of chyluria more or less persistently, and has not been in the West Indies for seven years.

Dr. Lewis says: "Not only may these hæmatozoa found in man live for a period of more than three years, but there is no evidence that they have any tendency to develop beyond a certain stage as long as they remain in the circulation."

Toward the end of April, our patient complained of pain in the right iliac region, and there was some indistinct swelling there. In the beginning of May came a lump, just below Poupart's ligament, in the right thigh, consisting of several enlarged lymphatic glands. This swelling was very painful, and the resident physician thought it enlarged at night and decreased by day. About this time filariæ were very scarce in the blood, many slides being examined without finding any. It was hoped that the parent worm might have lodged at this place, and that if it were removed the symptoms would cease.

May 13th, the bunch of enlarged glands were dissected out; during the operation several white filaments, not adherent to the surrounding tissue, were encountered; one of these, about the diameter of a hair, was drawn out, from the gland tissue apparently, for about three inches, and as it could not be drawn further and was quite tough, it was cut off and at once examined under the microscope by Dr. Peabody, who considered it nerve tissue; it was proposed to examine this more carefully at leisure, but unfortunately the specimen was lost.

Although we were not sure of having removed any parent worm, during the two months of the patient's sojourn in the hospital subsequent to the operation, no more filariæ were found in his blood, although it was examined frequently and at all times of day. He said he felt better and stronger, and his urine became clearer, some days appearing quite normal, although at other times it was as milky as ever, without discoverable cause.[1]

POSTSCRIPT BY THE EDITOR.—Through the kindness of Dr. Peabody, pathologist to the New York Hospital, by Dr. Hackley's permission a number of physicians were on different occasions allowed to observe the filariæ in this case.

On April 26, 1882, about midnight, Drs. Peabody, Amidon, and myself, made the following observations. In the two or three drops of blood procured by Dr. Peabody we found very few filariæ, but we were able to study them quite carefully with several good objectives, giving from one hundred to five hundred diameters. We were particularly interested in the appearance of the extremities of the parasite, from which we all saw, or thought we saw, filaments project. These were excessively delicate, tapering, and whip-like, the one attached to the tail being considerably longer than that at the cephalic extremity. We all noticed sudden movements of those whip-like filaments, which seemed to me movements of protrusion. This was more especially evident at the cephalic end of the filaria, where careful observation with five hundred diameters, by a good lens, revealed a sharply marked head, with an approach to a mouth or sucker-like termination, out of which the filament presented, and from within which it seemed to suddenly project and recede again.

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[1] The editor is informed at date of proof-reading (Sept. 19), that the patient is in the Presbyterian Hospital, and that his blood contains filariæ again.

At no time did this filament, at either end of the filaria, present flat, or, with double contour, or wrinkled, like the sheath-like projections figured in various works,[1] or as seen by Dr. Belfield in the filaria of Dr. Mackenzie's patient in the London Hospital. Some observers have seen this membrane at both extremities of the filaria; others, only at its tail.

In order that both conceptions of the nature of these appendages may be laid before the readers of the ARCHIVES, I have had two wood-cuts made. Figure 1, represents the view generally adopted, that there is a membranous sheath within which the filaria moves to and fro; this was made from a photograph of the dead and stained parasite in blood taken from Dr. Mackenzie's patient, London, last spring, by Prof. Wm. T. Belfield, of Chicago. I wish to take this opportunity to thank Dr. Belfield for his permission to make use of his photograph. The second figure represents faithfully what we saw at the New York Hospital laboratory on April 26th. The worm was carefully measured in breadth and length, and the figure is just as it appeared, magnified five hundred diameters. The delicacy and length of the filaments are also approximately exact.

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[1] Perls: "Lehrbuch der Allgemeinen Pathologie," ii Theil 1879, p. 65.
"Verhandlung des Vereins für innere Medicin," Berlin, Jahrgang i, 1881-2, pp. 105-106.
Virchow's Archiv, Bd. lxxxix, Heft 2, p. 365, August, 1882.

ON A PECULIAR CUTANEOUS LESION (ULCUS ELEVATUM) OCCURRING DURING THE USE OF BROMIDE OF POTASSIUM.

By E. C. SEGUIN, M.D.

IN the last few years, more especially since the publication of Trousseau's lecture entitled "*Exanthèmes sudoraux*,"[1] much attention has been paid to toxic or medicamentous eruptions.

Among these, the cutaneous lesions produced by the various bromides, when taken internally, have been particularly well studied, and quite a variety of eruptions have been observed and recorded by dermatologists and neurologists.

In the last edition of Prof. Duhring's excellent work[2] we find a section devoted to this subject, and the following bromic eruptions are described under the head of *dermatitis medicamentosa.*

1. Acne-form pustules.
2. Brownish discoloration of the skin.
3. Simple papular eruption.
4. Confluent or molluscoid acne.
5. Maculo-papules.
6. Carbuncular acne.
7. Bullæ.
8. Rupia.

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[1] *Clinique médicale de l' Hotel-Dieu*, 2me éd., 1865, i, p. 199.

[2] "Practical Treatise on Diseases of the Skin," 3d ed., Phila., 1882, p. 348.

Ulcers are not named in this list, and I do not know that they have ever been described; unless it be in the shape of isolated ulcerated carbuncular acne.

It has been my fortune to observe this year two cases of large, elevated ulcers upon the legs, occurring in epileptic patients using the bromide treatment, and I desire to place on record this new form of cutaneous lesion, probably medicamentous in origin.

Case i.—Miss C., aged 25 years, epileptic since her fourteenth year. The disease has been fairly well controlled by moderate doses of various bromides, under the supervision, in the last few years, of her physician, Dr. P. C. Barker of Morristown, N. J. Very little facial acne has appeared, and at no time has severe bromism been apparent. Some ten years ago, while the patient was in Germany with her family, there appeared some "boil-like" sores upon the lower part of each leg, which soon coalesced into ulcers, one on each leg, presenting very much the same appearance as those of later development about to be described. On the left leg there was an "ulcer," also below the head of the fibula. In a few months these ulcers healed. So far as the mother of Miss C. knows, there was no increase in the amount of bromides, or change of bromide, prior to the appearance of the eruption, nor does she remember any special treatment, other than the application of some herbs to the sores, which determined their healing. The patient, continuing the bromide (various formulæ) treatment steadily, had no further serious eruption or ulceration, indeed no special bromic symptoms, until about fifteen months ago, when large, purplish papules, like "boils," appeared upon the outer side of each leg, about four inches above the external malleolus. These discharged, leaving ulcerated spots which coalesced, forming an ulcer which increased quite steadily in size until the early spring of this year, when I saw the patient. This account of the early appearances and development of the sores is derived from the patient's mother, whose qualifications as an observer are fair. Still, the lack of professional study of the early stages of the lesion is a *hiatus* much to be regretted. It is doubtful if the ulcer was ever a common excavated one; Mrs. C. thinks that for months prior to my examination its surface was distinctly raised above the level of the healthy skin.

When I saw Miss C., in March of this year, there was a large ulcer on each leg, almost encircling it, leaving a bridge of healthy skin over the anterior surface of the tibia. The rest of the leg, at a level about four inches above the malleoli, was covered by an irregular ulcerated patch, raised from 2 mm. to 4 mm. above the skin. Its area was irregular, somewhat pear-shaped, with its largest development posteriorly. Its greatest width, vertically measured, was perhaps 8 cent. The surface of this "ulcer" was different from any thing I had ever seen. As stated above it was raised quite uniformly above the healthy skin ; its edges were abrupt, almost vertical, and showed no signs of cicatricial action. The elevated floor of the ulcer was firm, grayish-red in color, with here and there an adherent crust ; it secreted a fetid, sanious, puriform liquid, and bled upon being touched with moderate violence. It did not look like ordinary granulation tissue ; it was much firmer, composed of larger masses, and, in fact, at several points it presented a slightly villous or rather papillomatous appearance. There was no burrowing of pus under the ulcer, and the rest of the leg was normal,—perfectly free from "boils" or papules. This description, insufficient as I feel it to be, applies to both legs ; the ulcers upon them were almost precisely alike ; the lesion was a symmetrical one. (On the left leg, just below the head of the fibula, was an irregularly-shaped, smooth, white and coppery cicatrix of the ulcer which appeared ten years ago, and healed in a few months.) The patient's face and neck presented hardly a trace of bromic acne, and I was told that her body was free from ulcers or other eruptions ; she showed none of the other symptoms of bromism, and her epileptic attacks were still recurring in a mild form occasionally.

I was so much impressed with the firmness and elevation of these patches that I could not avoid a suspicion of epithelioma, in spite of the symmetry of the lesion which indicated its toxic or systemic origin. Prof. Henry B. Sands, to whom I then sent Miss C., decided that there was nothing malignant about the ulcers, and he looked upon them as eruptions connected with the patient's condition or with her medicines, and he sent her to Prof. William H. Draper, who also looked upon it as medicamentous eruption. Dr. Draper has recently written me as follows concerning the case :

". . . I remember Miss C.'s case perfectly, even though I have no notes of it. . . . I examined some scrapings from the ulcer and found only granulation cells. I think it was unquestionably a bromic acne. The lesion begins, I believe in the follicular structures, it is said in the sweat follicles, but I suspect in the sebaceous ones as well. . . ."

Acting upon Dr. Draper's suggestions, Dr. Barker applied pyroligneous acid to the ulcers and they at once began healing. I saw the patient a fortnight ago and found the ulcers amost entirely healed, with copperish smooth cicatrices such as we see after common ulcers of the legs.

It is interesting to note that the bromide treatment was continued unchanged.

Case 2.—Annie L., aged twelve years was brought to me at the Manhattan Eye and Ear Hospital on February 24, 1882, to be treated for peculiar attacks which I considered epileptic, and of a form intermediate between petit-mal and psychical epilepsy. In many seizures there were hallucinations of vision. The first attacks, which appeared about eighteen months ago, after a fever, were distinctly spasmodic. In the last few months more attacks of both sorts; right internal strabismus has appeared. Much complaint of headache, principally over the right eye; grasp of right hand less strong than that of left. Further details concerning the neurosis are unnecessary for my present purpose.

The child's health seemed good; she was fairly well-colored, and her skin was everywhere normal.

She was put upon our regular bromide treatment, taking from three to four grammes a day at various times. The indications of coarse cerebral disease were so strong that I also gave her .75 grammes of iodide of potassium three times a day, and applied a few blisters behind the ears.

The epileptic attacks were at once controlled, and the child seemed to bear the medicines well. Owing to some annoyance at having to wait very long one day, the mother ceased bringing the child to the hospital; this was some time in March, and there was then no bromism or eruption.

Rather accidentally, Annie again came under my care in June, by being brought to my clinic at the College of Physicians and Surgeons. I at once recognized her, and upon inquiry found that she had had private treatment since leaving the hospital, and had taken bromide of potassium only (no iodide). The attacks had been few and slight, more of the psychic order: fear of falling down stairs, seeing animals, weeping, calling out to mother, and complete amnesia of attack. She was somewhat stupid from the bromide, but the amount given was impossible to ascertain. There was almost no facial acne; the child's color was clear and healthy.

The mother, however, stated that since leaving the hospital clinic, the child's legs had become the seat of very painful sores, which she wished me particularly to examine. Bandages were removed from both the child's legs, and my surprise may be imagined when I beheld ulcers precisely like those of Miss C., seen a few months before.

The lower part of each leg, some 5 cent. above the malleoli, was encircled by a large ulcerated patch, distinctly elevated above the surrounding healthy skin. The outline of the sores was very irregular, varying in vertical width from 3 to 8 cent.; the largest surface of each sore being on the fibular side of the leg. The edges were sharply defined and nearly vertical. The surface of the sores, raised 2 or 3 mm. above the healthy skin, was covered with brownish-black scabs and a most offensive sanies. On removing some of these scabs a rough granulating surface, easily bleeding, was revealed. I use the word granulating, but the appearance was that of a firmer, more villous, in places almost papillomatous formation, than the delicate translucent and uniform surface of ordinary granulations.

The mother, a not over-intelligent Irish woman, stated that this local trouble began in April, while using the bromide prescribed by the physician she consulted after giving up the hospital. At first the right leg was affected with large pimples or boils, which "broke," ulcerated, and coalesced into an open sore. In about a fortnight the left leg was similarly affected. It is interesting to note that this woman's account of the beginning of the ulcers agrees precisely with that given by the very intelligent mother of Miss C.

I directed that the ulcers be gradually cleaned of scabs by frequent washing, and that a strongly carbolized ointment be used twice a day. The dose of bromide of potassium was fixed at two grammes night and morning. The mother, having other children to attend to in the midst of the difficulties of tenement-house life, did very little toward cleansing the sores, and did not apply the ointment as carefully as necessary. Besides, the child dreaded to have the legs dressed, and cried violently each time they were washed. I was away a great part of the time in the months of July and August, and during that time there was no material change in the size or appearance of the ulcers. The child remained free from ordinary bromism, and her complexion was good. The seizures occurred but rarely.

On July 28th I gave her ether, and after removing nearly all the scabs from the left ulcer, I applied to it Paquelin's cautery quite freely, and ordered a carbolized lotion for a few days, to be followed by applications of balsam of Peru ointment. At the same time I cut out a strip of the ulcer and adjacent sound skin for histological study ; this was pinned on a cork and placed in bichromate of potassium solution. In cutting away this little flap I demonstrated that there was no appreciable lesion of the subcutaneous connective tissue ; the derma seemed hypertrophied, but the whole piece was easily dissected away with the scalpel. I reduced the bromide one gramme night and morning, and gave five drops of Fowler's solution after each meal.

I saw the child again Aug. 21st. The ulcer which I had cauterized, that on the left leg, was nearly half healed over ; the right leg presented the same appearance as at first, and in spite of my urgent orders had not been well cleaned of scabs and sanies. I directed that to be done before the child was brought again, intending to use the cautery again, or to apply nitric acid. The same amount of bromide was to be taken night and morning, and eight drops of Fowler's solution were to be given after each meal.

I have met with a third case, in which the legs were affected in a way not unlike what the mothers of Cases 1 and 2 describe as the first or carbuncular stage of the ulcer; and probably, if the bromide treatment had been persevered with, ulcers might have formed in this case also.

Miss A. B., aged 17 years, a handsome girl, with a very fresh complexion, of German parentage, consulted me on November 1, 1880, for epileptiform attacks. She was a healthy child. At 8 years had an attack of typhoid fever lasting several weeks. On getting out of bed at the end of eleven weeks, found that she could not walk. It was nearly spring (fever in August) before she walked freely. Arms were unaffected ; mind normal. In her ninth year had a convulsion one morning, biting her tongue. During the day legs became quite weak, especially the right. There seemed well-marked paralysis, but it is not certain whether there was any muscular wasting. Under galvanism and strychnia she gradually regained the use of her legs, but ever since she has had convulsions at very irregular intervals—every two or three weeks, or at interval of months. In the last two years three or four severe attacks, last one in September. Has had occasional doses of bromide, but no systematic treatment. Patient states that in night attacks she wakes dizzy, has time to call some one, hears a loud noise as of a wheel going faster and faster, until she loses consciousness; hears no voices or bell-sounds. A cousin who has witnessed seizures, says that that there is a severe convulsion, in which patient's eyes are open; after attack, she is stupid, and wants to rise; talks and weeps violently. Then she falls into a heavy sleep.

Last summer, was exposed to severe solar heat and had a profuse nose-bleed. Ever since has been liable to petit-mal; a whirling dizziness, followed by "faintings" in some instances. Further questioning shows that ever since first spasm she has had a third sort of attack, consisting in the sudden appearance of "balls before the eyes," followed by temporary diplopia; no drowsiness.

In 1877 Dr. Brown-Séquard was consulted, and gave Miss B. his bromide mixture. This was faithfully employed for six months. The patient then had large sores with scabs upon her legs, mostly on right. It is difficult to ascertain whether these sores were like those described above, or discrete scabbed sores, such as I have seen in other patients and termed rupia-like. There was no facial acne, and the epileptiform attacks were suspended. The bromides were stopped and the sores quickly healed.

Examination showed some anæmia and symptoms of gastric catarrh. The case was so clearly epileptic, that in spite of the former bad effects of bromides, I persuaded the patient to try them again; giving her my solution of chloral and bromide,[1] three teaspoonfuls (twelve grammes) at bedtime, well diluted. Treatment and diet were also ordered for the gastric catarrh. In eight days a "boil" appeared on the right leg, constituting a small abscess, which ruptured spontaneously on the twelfth day, near a scar of the former eruption three years ago. Dose of bromide solution reduced to two teaspoonfuls at night. On the fifteenth day several large indurated pimples had appeared around the small abscess. On the nineteenth there was quite a crop of large, purplish papules with evident tendency to suppuration on the right anterior region. This appearance, the patient said, was identical with that observed while using the Brown-Séquard mixture three years ago, and she begged me to stop the bromide. I might add that for a week or ten days arsenic and sulphide of calcium had been given to control the eruption. There was no bromism, hardly a trace of common facial acne, and the gastric symptoms were better.

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[1] Chloral hydrate, 15.; potassium bromide, 30.; aqua, 200. grammes.

I suspended the use of ordinary bromides, and gave bromide of zinc, bromide of camphor, digitalis, valerian, etc., at different times; also, at times, renewed treatment for the dyspepsia, and tonics. The epileptiform attacks have been almost perfectly under control, and there has been no return of the eruption (which passed away in a week or ten days after the bromides were withheld).

Very probably, had I persevered in giving the solution of bromides, the papules would have all undergone suppuration and ulceration, coalesced, and formed an ulcer more or less like those observed in the other cases.

This third case, though incomplete, has this value, that it bears out what the mothers of Cases 1 and 2 claimed as to the early appearances of the ulcers, viz.: at first an acne, undergoing suppuration and ulceration; the resulting ulcers merging into one sore.

The following points in the clinical history of these ulcers are interesting:

1. Their origin in acne.
2. Their progressive and semi-malignant tendency.
3. The absence of bromism in the patient.
4. The absence or slight development of common facial acne at the same time.
5. The possibility of curing them by energetic local treatment (Case 1) without omitting the bromides.
6. The inefficiency (?) of arsenic and of calcium sulphide (Case 3).

Histology.—Sections of the piece of tissue removed from the ulcer show great increase in the thickness of the rete Malpighii, with hypertrophy of the whole skin in places. In several places villosities visible to the naked eye occur, made up of all the elements of the skin thrown up and out into a minute mushroom-like or polypoid mass. In other places proliferation of young cells has taken place in the cutis, with atrophy and rupture of the epidermal layer, and partial escape of the newly-formed tissue, constituting a sort of abscess, opening externally. In other localities the patches of inflammation were wholly circumscribed, and subepithelial. The deeper layers of the skin, and to a certain extent the subjacent connective tissue, are infiltrated at certain points with young cells. The papillæ, hair-follicles, and sweat-glands do not appear to be the seat of any primary or important inflammatory change. In no part of the section was its surface (edge) covered by granulation tissue, as in a common ulcer.

From these appearances we may conclude that the ulcer resulted from a dermatitis, which was partly suppurative, but largely hypertrophic.

EDITORIAL DEPARTMENT.

POST-GRADUATE INSTRUCTION.

A number of physicians of this city, mostly specialists, of conspicuous disparity in reputation as men of science or as teachers, have formed themselves into a faculty of medicine, and propose to organize a college for the purpose of teaching certain things, mostly matters which come within the limits of specialties, to graduates in medicine. So sanguine are these gentlemen of success, and so confident of the righteousness of their cause, that at least one of them has appended his new professional title to his name, on the title-page of a book—— before the college has been opened and before one lecture has been delivered.

There is something captivating in the term post-graduate instruction, or post-graduate course, and something imposing in post-graduate faculty ; and very naturally one interested in medical education is at once led to inquire whether there is any need of post-graduate instruction as such (apart from that never-ending effort at self-instruction which intelligent and conscientious physicians make through their lives), and whether the proposed new faculty is indispensable for the purpose, or whether an even better course of advanced teaching for the senior medical student could not be organized in the colleges already existing, and graduates invited to follow this part of the curriculum.

The first question, is there any need of post-graduate instruction ? can, I regret to say, be answered only in the affirmative. Taking the men who are yearly sent out of our medical colleges with diplomas, we find that in spite of all they have acquired at the hands of their alma mater they are in need of more teaching, at least of more practical, educating teaching. A proof of this statement lies patent to all, in the fact that the very best men of these medical classes, those who have passed the most brilliant examinations and shown that they have crammed successfully, the very honor and prizemen of the schools, immediately compete for hospital appointments which will make them pupils again; they will give themselves another and a better chance at self-improvement. This is a tacit acknowledgment of the incompleteness of their college education; and if these well-crammed young men feel this way, and admit as much, what must be the condition of the rest of the hosts of graduates who, besides lacking practical education and training, are less well provided with memorized knowledge. They all need post-graduate instruction, and many of them will always need it and be glad to come for a few months to a large city to attempt to do what they ought to have done, or, more justly, ought to have been enabled and obliged by their alma mater to do before receiving a diploma. It is from this very numerous class of graduates of imperfect medical schools, who are men of all ages, of various degrees of practical success, but of one mind as to their need of better training, that the proposed post-graduate medical school is to be supplied with pupils or followers.

At the present time each medical college in our great cities bears upon its matriculation lists the names of many such seekers after scientific improvement,—men who need training and special teaching. They find themselves lost in a crowd of youths who are struggling to learn enough by rote, through books and lectures, to pass examinations of greater or lesser severity. The graduates find the lectures, as a rule, to be a mere rehash of what is in the books, made for the purposes of keeping up the time-honored plan of lecturing to large classes, and to give the student a sort of repetition of the various subjects. The impressions gathered through two senses, reading the matter by one's self and receiving it through the ear in an amphitheatre, are supposed to be more firmly fixed, better registered, and more definitely associated in the brain, than they would be if derived only through reading. That there is a degree of psychological truth in this no one will deny, but the crying evil of routine didactic teaching cannot be sustained on such a plea. Besides hearing lectures which are repetitions of chapters of books which he knows well enough, and therefore useless to him, the graduate finds no lectures given by the only men about the schools who could teach him something new and vital, viz.: the specialists. They are, as a rule, not allowed to lecture in the regular course of lectures, and are placed in charge of clinics.

To these the graduate goes only to be disappointed and hindered in his work of self-improvement. He finds most clinics conducted without the least system, being merely an exhibition of cases as they happen to present themselves, with remarks by the professor. In many clinics, extraordinary cases of very little importance to the physician are made to play a great rôle, consuming time which should be devoted to the practical learning of methods of diagnosis and therapeutics, and to the systematic study of common diseases. Lastly, the graduate finds the front seats of lecture-rooms at the clinics crowded by junior students, who are there without appointed tasks, and who come to the clinics partly from curiosity to see disease, and partly because they have a dim notion that they must get all the "clinical experience" they can, even in their first years. The machinery of most of our schools is as yet so inadequate that there is no means of excluding the junior student from lectures and clinics which can do him no good, which use up time which should be devoted to a study of the fundamental branches, and where he occupies a seat which the undergraduate or graduate has a moral right to.

These difficulties dishearten the practitioner who has come to a medical centre, perhaps at much expense and inconvenience, to improve his scientific and practical knowledge.

But some say to him: Why do you not take advantage of the numerous private classes which are organized during the winter session, either for cramming purposes, or to teach special subjects in a practical way? This our practitioner does, but he finds it quite expensive, and also has to encounter another difficulty, similar to that which he found in the schools: the classes are organized more for the teaching of undergraduates, by very elementary methods; he does learn something, but not as much as he hopes to, and not exactly what he needs. The proposed post-graduate school will say to the numerous graduates who return to New York for self-improvement: Come to us, gentlemen; we offer a curriculum prepared specially for you; you will not be elbowed by a crowd of raw students; we will give you every desired opportunity to study in a scientific and practical manner.

It remains to be seen whether the proposed new school of medicine will succeed, by such an appeal, in attracting a respectable number of pupils.

This brings us naturally to the consideration of the second proposition, whether a better course of advanced teaching than this new faculty offers, for senior students and for graduates, could not be organized in the already existing medical schools, as a part of their curriculum.

The qualifications of the members of the new-born faculty, and the discouraging circumstances they will have to contend against, are negative points in the discussion of this question, which we do not care, and do not need, to take up.

On the other hand, the advantages which the existing medical colleges have for undertaking higher teaching are very great.

Most of their professors, and nearly all of their assistants, are connected with hospital and dispensary services, and thus the colleges can command almost unlimited clinical opportunities. These means of teaching are now allowed to go to waste, chiefly because the faculties take no pains to bring these various services into a logical system. All the men connected with the school, and who have clinical services, teach as they please, several of them going over the same ground for the same unwieldy class of students. There is no attempt made to subordinate and correlate the various clinics in the college, in hospitals, and at dispensaries, to the plan of teaching at the college, and to certain series of didactic lectures. Indeed, it seems to be nobody's business, and "go-as-you-please" appears to be the watchword among medical teachers. An enormous amount of clinical material is thus allowed to be ill-used or not used at all.

It seems to us that, taking advantage of the disinterested desire to teach others which the talented young alumni of a medical school always manifest, and of their interested, though laudable desire to acquire a nominal connection with their alma mater, much good clinical teaching might be secured without any great pecuniary outlay; only at the cost of careful planning of a scheme, and of energetic supervision of its execution.

With each chair upon a practical branch (each elementary scientific branch, chemistry, anatomy, physiology, and materia media, should have its laboratory with competent teachers), quite a number of younger men might be associated with some such title as clinical instructor or clinical professor, according to the professional standing of the incumbent. This title, desirable in itself, should convey with it to each appointee the various obligations to use his hospital or dispensary service mainly for the advantage of the school, to teach a number of months in each year to limited classes provided for him by the secretary of the faculty of his school; to regulate his teaching in all reasonable ways (matters of scientific opinion excepted), according to the suggestions of the professor of his branch at the school, who would thus, besides delivering lectures and giving clinical instruction himself, control and direct a large and highly efficient teaching force and a vast clinical material. By such means the senior student, or under-graduate, and the graduate in search of further teaching, could be registered and distributed in classes, to their great advantage. On the other hand, the school would have this reward, among others, that between these numerous junior teachers the liveliest rivalry would arise as to excellence in teaching and faithfulness in serving the school, and thus there would grow up a strong competing class of candidates for any vacant professorship.

It may be said that these clinical teachers would demand emoluments. We doubt it, partly because it is in the nature of young men (young physicians perhaps more especially) to labor for the sake of an honorable title, of a possible promotion to an assured position, and I place it last, but not least, disinterestedly for science's sake.

This relates to teaching of the practical branches at the bedside, without the school principally.

Now, what need the schools do to improve higher teaching within the college walls, and to offer a post-graduate curriculum better than that planned by the proposed school?

The thorough discussion of such a question would involve a consideration of the means of improving teaching for all grades of students; a much too complicated theme. We must content ourselves with suggesting some simple changes in the present arrangement which would, in our opinion, fairly meet the wants of students about to graduate and of graduates seeking higher instruction.

In the first place it would be necessary to break through the present rule in most medical colleges, that only one lecture or clinic shall be offered to the class at one time. It is difficult to find polite terms in which to condemn this time-honored (American) custom, so contrary does it seem to common-sense and to economy in the administration of a school. Here is a college building costing from forty to sixty thousand dollars, with a full staff of professors and assistants, with a class of three or four hundred students, and forsooth the whole of this machinery must result in but one form of teaching at any one time, for a few hours a day. This is comparable to an attempt to irrigate a field from an ample reservoir, but the water must be made to flow through one little aperture; the area of the field and its need of water on the one hand, and the abundant supply of water on the other hand, are made subject to a meagreness of distribution which is justified only by a senile custom. No doubt this practice had its rise in connection with the now antiquated notion that medicine was to be taught didactically, and the whole class obliged to hear the various lectures systematically. And, in the same way, the class was supposed to attend every clinic. By all this, wholesale superficial education was reduced to a system and made easy. The truth is that from three to six lectures, clinics, or private classes might be going on at the same time, during the same hour, if the building were appropriate, and if an official had the responsible duty of classifying students and distributing them in classes.

As regards buildings: Our medical schools (in New York) have, as a rule, only two huge lecture-rooms or amphitheatres, and one dissecting-room. Even with this insufficient number of rooms, the present amount of teaching might be doubled. But in some schools more room might be had by devoting to a useful purpose rooms which are occupied by professors for a few minutes each day, merely as a convenience. Out of several professor's rooms one or more small lecture-rooms might be arranged at small expense. Besides, some of the schools could well afford to hire or purchase a private house near the school, alter it somewhat, and thus obtain two or three small lecture-rooms, and two or three laboratories.

As regards the arrangement of studies and classes: A thorough system of graded study by divided classes may be desirable, but we are hardly ready for this. No one, however, can claim that the time has not arrived for a strict division of our large classes into junior and senior classes, and to these might be added a graduate class. These groupings should be formally made by registration in the books of the official in charge of the college work, and by means of tickets and door-keepers the separation should be practically enforced.

By such means the junior students would be obliged to give all their time to the lectures and demonstrations upon anatomy, physiology, chemistry, and materia medica. They would also find time for special practical laboratory work in all of these branches, in the smaller teaching rooms of the school; and some of the more capable members of this class would find time to study also German or French, and perhaps medical physics and botany, branches which are almost necessary to a *complete* medical education.

As matters stand now, except in the case of the well-advised and seriously-minded few, junior students wander into clinics and hospitals, anxious to behold the horrors of disease, to experience the new sensations produced by the sight of a bloody operation, and, truth compels me to add, sometimes to gratify a lewd fancy by witnessing exposures of the human form. These young men have nothing to gain by going to clinics and visiting hospitals; they waste their time, of which they have only too little to do justice to the noble sciences they need to study; they take up room which the senior student or graduate should have. We are sure that many clinics are thus made to appear crowded and astonishingly successful; yet we hope and believe that the professors of such clinics would much rather teach a selected smaller class, free from the throng of curiosity and sensation seekers. The faculty of a medical school owes it to these misguided junior students, owes it to the seriously studious senior student, and more particularly to the earnest graduate who comes to the school seeking after higher instruction, to police the class, and by all the means in its power to restrict the junior class to its legitimate studies, and to keep it out of the rooms or hospitals where the practical branches are taught.

The senior or undergraduate class, studying more particularly pathology, practical medicine, surgery, obstetrics and gynecology, and therapeutics (that ill-taught though crowning science), might continue to hear didactic lectures, since it is premature to ask the partial or complete omissions of these; but the extensive clinical scheme proposed in the first part of this paper, should be employed as the chief means of education. The class could be subdivided into sections, and distributed in such a way as to allow each student an opportunity of practically studying disease in its various forms, of acquiring the arts of diagnosis and of treatment. It would be more especially for this purpose that the utility of numerous rooms for teaching would appear. Why, for example, should not, from 10 to 11 A.M., the junior class listen to a lecture, a demonstrative lecture let us hope, upon anatomy in one amphitheatre, while the senior class is hearing a lecture upon a surgical topic, and a selected section or two are doing laboratory work of some sort? Why in the afternoon should not sections of the senior class be at two or three hospitals attending bedside teaching or operations, and other sections busy in the same way in the college itself, or doing laboratory work?

The post-graduate class might well find opportunities for higher study in these small, well-officered clinics. This teaching, while not particularly scientific, would be practical, and would be better than any thing offered them in their student days. But much more might be done to meet the wants of the graduate class without interrupting the regular work of the school, with very little, if any, expense to the faculties, and we believe it might be done much better in the existing schools by their own teachers, than outside in a new special post-graduate school by a fortuitously conglomerated faculty.

The graduate in search of more instruction wants above all to get the opinions and newest practical ideas of specialists—of specialists in *study* if not close specialists in *practice.* He also wants (perhaps *needs* it more than he *wishes* it) instruction in methods: the education of his eyes, ears, and hands to *do*, to *execute* the things of science. He needs to be taught to manipulate all instruments, and to examine the human body thoroughly. He needs to learn practical therapeutics, including the use of electricity, hydrotherapy, massage, which are arts in themselves.

The rooms which should be added to all our present medical schools could be utilized for the teaching of these practical specialties. Lectures, clinical or demonstrative ones we mean, upon these subjects might be going on at the very same time that the regular school lectures were being delivered and clinics held. Each school has affiliated with it a sufficient number of younger specialists, or quasi-specialists, who would gladly, with a nominal honorarium or even without any, give the instruction in such a post-graduate course. Every specialty could be represented by one professor and by several clinics in hospitals or dispensaries, and members of the graduate class assigned to these, in accordance with their needs and wishes, by the secretary or registrar of the school. The classes which would attend such very special lectures, lectures embodying the freshest and most applicable science of the day, might be small, but their members would be seriously studious or even enthusiastic.

A post-graduate faculty organized in this way, from the younger men growing up around our medical schools, would do much to increase the reputation of their alma mater as a centre of learning, and would probably be stimulated to do much for medical science by more original research than we have at present in America. Though perhaps lecturing at the same hour with the professors of the principal branches in the students' department, there would be no rivalry or interference, if the students were classified and kept classified as above suggested.

An objection to this plan of subdividing the classes, and having several lectures, clinics, or demonstrations going on at once, is that some members of the senior class need to go over part of the elementary branches before presenting themselves for their final examinations ; they should have an opportunity of attending the lectures upon these subjects, and in the scheme proposed they could not do this without missing some clinic or lecture upon a practical branch. The fallacy of this objection is obvious ; it is made by one who believes in the superior power of didactic lectures to instruct. The prestige of didactic lectures has been already much dimmed, and we may live to see them almost banished from schools. The objection is invalid, because an intelligent member of the senior class can review his anatomy, physiology, etc., from text-books (having already studied these branches thoroughly and practically for a year or two).

Another objection, more serious, is that a well-managed division of the class and the exercise of effectual control over the course of study followed by its members would throw a considerable additional labor and responsibility upon the faculty. True, but this would hold good of any serious scheme for improving medical education. A part of the complicated badness of our present way of teaching students, has been the irresponsible ease with which a few men have done the business. A few faculty meetings in the year, an hour's lecture three or four times a week, with one or two clinics each week (not prepared beforehand—mere off-hand conversations about cases), and a rather busy week at the time of the annual examinations,—these have constituted the labors of a professor in a medical school. One might say that it would not be very terrible to make an addition to these duties in the interest of the class, and of medical science, but the scheme does not call for this. It would, however, demand that the gentlemen composing our medical faculties, with a deeper sense of the great responsibility they assume in attempting to educate three or four hundred young men, should carefully select one of themselves, or an outsider, a younger and less busy man than any of them, to co-ordinate the scheme of instruction. To do it well would tax the whole energy and time of a man having special executive ability. He might be termed secretary or registrar, and his duties would embrace keeping classified lists of the students, divided into junior and senior classes, and subdivided into sections for assignment to the various teachers connected with the school. He would have to see the students and teachers frequently, and secure such a rotation of these various sections as to give every student a full opportunity to study all branches of medicine. The numerous clinics affiliated with the school, held in hospitals and dispensaries throughout the city, should be, not under his control in any scientific sense, but guided and co-ordinated by him, in reference to their order of working. He could also keep a record of the various examinations passed by students of the three classes, junior, undergraduate, and graduate.

Finally, graduates submitting to such a scheme of study, devoting several months to study under such favorable circumstances, would naturally expect a certificate of their attendance and attainments. The members of the special faculty might be empowered to examine gentlemen desiring such certificates, and they could be issued by the medical school or by the university with which it might be affiliated.

E. C. SEGUIN.

NEW BOOKS AND INSTRUMENTS.

Studies in Pathological Anatomy. By FRANCIS DELAFIELD, M.D., Adjunct Professor of Pathology and Practical Medicine, College of Physicians and Surgeons. Nos. 20, 21, 22, 23, and 24. New York; Wm. Wood & Co., 1882.

The figures indicated above are endorsed upon the last fasciculus which our author has given to the public, of his studies in pathological anatomy. It is difficult to perceive the *rationale* of the system of numbering applied in this case, for the whole fasciculus is devoted exclusively to the consideration of one matter—the pathological anatomy of acute phthisis. Just why five numbers have been printed upon the paper-cover of a contribution to a single subject, does not appear upon its face, and the explanation is not suggested by any thing contained within.

Beyond this criticism, perhaps unimportant, we have nought but terms of commendation of Dr. Delafield for the careful, patient, thorough, independent, and unbiased manner in which he has made this study, and presented the result of his own examination of a most difficult subject. He has succeeded in throwing much new light upon the labyrinth of misunderstanding and misinterpretation, and deserves, therefore, much praise at the hands of his co-laborers.

The importance of this subject, not less than the earnest and valuable work of the author in its elucidation, warrants the use of more space in the ARCHIVES, than a mere passing mention. We feel justified, therefore, in presenting to your readers a somewhat lengthy abstract of the opinions of this investigator, and the observations upon which they are based.

After briefly sketching the clinical history of acute phthisis, Dr. Delafield proceeds to depict the *post-mortem* appearances of the lungs. A considerable portion of one or both lungs is found to be consolidated. But the consolidation is not of uniform aspect; the greater portion of it resembles ordinary red hepatization, or is of a peculiar grayish color, different from the gray hepatization of lobar pneumonia. In this diffuse hepatization are other portions of solidified lung, of different appearance. They are of a white or yellow color, much denser than the surrounding hepatization, or they may be softened at their centres; they are of many sizes and shapes—the smallest hardly larger than an air-vesicle; the largest may comprise a considerable part of a lobe.

Many of the smallest of these whitish masses are scattered irregularly through the lung tissue, like miliary tubercles. They really are miliary tubercles. The large masses seem to be formed of smaller ones. If the patient live long enough, many of these masses, both large and small, will be found degenerated and softened.

The varied conditions of the lung after death from acute phthisis have been interpreted in very different ways.

Some look upon the whole process as one of ordinary inflammation, believing that subsequent degeneration of the products of inflammation produces all the lesions. Others opine that all the white or yellow masses are essentially tubercles, and are surrounded by ordinary pneumonic hepatization, which they have excited by local irritation. Still others regard the whole as a genuine combination, side by side, of pneumonia and miliary tubercles.

None of these views seem to the author to account for all the lesions. For him the pathological condition is really a complex one, nothing being gained by an effort to make it appear more simple than it is.

It is necessary to examine each of the different parts of the lesion in detail, and see what their real structure is.

I. *The diffuse hepatization* generally resembles red hepatization of lobar pneumonia, but the cut surface is smoother. It may present a peculiar gray lustre, sometimes seeming almost gelatinous. It does not appear to degenerate or soften, except immediately around some of the nodules. The blood-vessels in the walls of the vesicles are readily filled artificially. The vesicular walls, or partitions, are unchanged, save that they are overlaid with new epitheloid cells; and the vesicular cavities are filled, more or less completely, with the products of inflammation—such as pus cells, fibrillated fibrine, large epitheloid cells, minute shining granules, and a peculiar transparent, shining matter.

Some vesicles may be entirely filled with the granules, others with fibrine, others with epithelial cells, while others still contain granules and epithelium with pus cells.

These granules cannot be dissolved by acetic acid, liquor potassæ, or chloroform, but they can be stained by methyl violet or hæmatoxylin.

This diffuse hepatization seems to have no tendency to cheesy degeneration or necrosis. The inflammatory products in the vesicles may undergo fatty degeneration, but the vesicular walls remain intact and the blood-vessels pervious. Clinical experience and physical signs tell us that this diffuse hepatization is capable of resolution, and may entirely disappear. If chronic phthisis set in, however, this diffuse consolidation undergoes marked changes.

II. *The white or yellowish nodules*—even those which present the same appearance to the naked eye—are not all of the same nature. A definite conception of the relations of small bronchi to the air-vesicles is requisite to a proper understanding of their anatomy. None of the descriptions of authors concerning this, corresponds exactly with what we see in normal and diseased lungs. They all imply the existence of a set of tubes which branch, becoming smaller and more numerous until they finally terminate in several minute tubes, into which open the air-vesicles.

In truth, the bronchi enter the lobules most irregularly. Some enter at the ends nearest the roots of the lungs; others at the side of the lobules, into which they send branches at right angles.

The air-passages, or infundibula, are given off from the end of terminal bronchioles, or from the sides of small bronchi. They are really part of the vesicular system of the lung, and not of the bronchial system. Each lobule seems to consist principally of such air-passages. Neither the bronchi nor the air-vesicles proper constitute as large a part of the parenchyma of the lung as do the air-passages. The latter seem to be made up of a succession of large vesicles opening into one another, or of an irregular large canal formed of vesicles, into which other vesicles open from all sides.

The white or yellowish nodules of phthisis seem to be formed in air-vesicles, in single air-passages, or in groups of them, as well as in and around the bronchi. They may be classified as follows:

1. *Smallest nodules*, not larger than a single vesicle or air-passage, are, in the fresh lung, not easily distinguished from the diffuse hepatization which surrounds them. They consist of a single

air-vesicle or portion of an air-passage more or less filled with "tubercle tissue," or partly with this peculiar tissue and partly with epithelium, fibrine, and pus. The vesicular walls remain intact, the blood-vessels permeable. Often the surrounding air-vesicles and passages are filled with epithelial cells and fibrine. They then constitute a peculiar hepatization which may be regarded as partly tubercular, partly inflammatory.

2. In *larger nodules*, besides the appearances noted above, the walls of the vesicles are affected. They become split up and changed into tubercular tissue. As this process proceeds, the outline of the vesicles is lost, and the blood-vessels can no longer be injected. Such nodules are identical in structure with the miliary tubercles seen in acute miliary tuberculosis.

3. *Peribronchitic nodules* partly or completely surround a bronchus or an air-passage. In size and shape they vary with the bronchus and the number of surrounding vesicles involved.

When closely examined, these peribronchitic nodules do not all seem to possess the same minute structure.

a. Some are identical in formation with the ordinary nodules of broncho-pneumonia of children and adults.

b. Others consist of a bronchus filled with pus and epithelium, the walls being infiltrated with "tubercle tissue," and surrounded by a zone of air-vesicles filled with tubercles, and around this last still another zone of vesicles filled with epithelium, fibrine, and pus.

c. Other nodules are found of the same general constitution, but the vesicular walls in the peribronchitic mass cannot be distinguished, and the blood-vessels cannot be artificially filled. After the contents of the bronchus, its walls, and even some of the "tubercle tissue" undergo cheesy degeneration.

d. In still other peribronchitic nodules, the bronchus may contain inflammatory products and the walls remain unchanged, whilst around the bronchus the air-vesicles are filled with "tubercular tissue."

In all these varieties of peribronchitic nodules a large part of the consolidation is due to changes in the air-vesicles.

Two lesions affect the bronchi directly in some cases of phthisis.

1st. The walls of some large bronchi are infiltrated with "tubercle tissue," which undergoes cheesy degeneration. This may sometimes be so advanced and extensive that no structure can be recognized. The cavity of the bronchus is dilated and contains inflammatory products, also in a condition of cheesy degeneration, and the adjacent air-vesicles are often affected. This dilatation may be sufficient to form a cavity of some size, which may further enlarge by disintegration of the cheesy bronchial wall.

2d. There may be a general dilatation of most of the bronchi in a considerable portion of a lung, without any marked change in their walls, and with only a moderate amount of inflammatory product in their cavities. This general dilatation usually affects the medium-sized and smaller bronchi. The lung tissue between them is usually consolidated, and when cut the tissue appears honey-combed.

The larger nodules of acute phthisis seem to be of three kinds:

1st. Peribronchitic nodules, such as described.

2d. Bodies resembling miliary tubercles in other structures, but much larger,—the "tubercules massifs" of French writers, which may suffer cheesy degeneration at the centre.

3d. Nodules consisting of a number of air-vesicles in a state of coagulation-necrosis.

Cheesy degeneration (Virchow) is recognized to be a transformation of the different elements, both the old and the new, of a tissue into an amorphous mass, composed of granular matter, fat and shrivelled tissue, the result of a process of death disintegration, fatty degeneration, and desiccation, whereby is formed a totally anæmic, dry, dense, and almost amorphous mass.

Coagulation-necrosis (Cohnheim) is a term having a meaning altogether different. It occurs in infarctions where the nutrient terminal artery is plugged or obstructed. Cheesy degeneration, however, may set in at the centre of an infarct and extend so as to involve the greater part of it. Portions of tissue, the seat of coagulation-necrosis, may resemble very closely areas of tissue which have undergone cheesy degeneration. It is in the study of phthisis especially that this similarity has led to confusion.

In acute phthisis, the nodules due to such a process of coagulation-necrosis are regularly present and form a very important part of the lesions.

There are two forms of coagulation-necrosis nodules. In each there is a centre of air-vesicles filled with inflammatory products in a condition of coagulation-necrosis; and around such a centre there may be merely a zone of ordinary hepatization, or there may be a zone of "tubercle tissue."

In these areas of coagulation-necrosis the blood-supply is cut off. The vessels are stopped. The outline of the vesicular wall is still visible, but is recognizable mainly by the elastic fibres existing therein. The vesicular cavities are filled with polygonal cells and shrivelled nuclei. Sometimes they are filled with coagulated fibrine only. The latter are not so opaque as the others, and they give to the cut surface of the infarct a mottled aspect. There is no gradual transition between the necrotic centre and the surrounding zone of hepatization.

Cheesy degeneration may begin at the centre of the necrotic portion, and extend out into the zone of hepatization or of "tubercle tissue." In such cases it is often difficult to distinguish these nodules from the peribronchitic nodules, or from the "tubercules massifs."

Through softening of these nodules, cavities of various size and shape may be formed. In this way sometimes a large portion of lung becomes riddled by small cavities having ragged walls, the number of cavities corresponding with the number of areas of coagulation-necrosis.

The only way to account for the formation of these centres of coagulation-necrosis, is by supposing an occlusion of branches of the pulmonary artery. The lesion is quite similar to that of the white infarction of the kidney or of the spleen. It has been demonstrated by different observers that the inner coat of the arteries is often altered in phthisis. The author has several times seen an obstruction or obliterating endo-arteritis of branches of the pulmonary artery in cases of acute phthisis accompanied by coagulation-necrosis. The various sizes of these necrotic areas would seem to be related to the blocking up of smaller or larger arteries.

In acute phthisis, therefore, we have lesions of peculiar character. The disease is very far from being a simple broncho-pneumonia with degenerative changes, or an acute miliary tuberculosis combined with pneumonia.

The prognosis seems to depend mainly upon the number and size of the areas of coagulation-necrosis, and it seems probable that most of these are formed at the outset of the disease.

The diffuse pneumonic consolidation may undergo resolution. The miliary tubercles and peribronchitic nodules do not occupy much of the lung, and may be transformed into cheesy material or fibrous tissue. But the areas of coagulation-necrosis destroy a considerable part of the parenchyma of the lung, and have a natural tendency to soften and form cavities.

The "tubercle tissue" referred to by the author is regarded by him as a delicate basement-substance more or less perfectly reticulated, provided with nuclei, and often enclosing spaces containing very large polygonal branched cells with many nuclei—giant-cells,—the branches frequently being traceable into connection with the reticulated basement-substance.

The subject-matter above treated of is, as usual, most abundantly and superbly illustrated, there being thirty most exquisite photographs and drawings reproduced, not a single one of inferior execution. At the end of the explanatory text follow an explanation of the photos, plate i–xciii inclusive, and a general index.

The present fasciculus, now passed in review, completes the first volume of this important work of Dr. Delafield, a monument of industrious and intelligent labor of which he can be justly proud. As the several fasciculi appeared from time to time, we had occasion, in previous issues of the ARCHIVES, to call attention to their merits and to their defects. We have before emphasized the value of the elaborate and costly illustrations, which, with a few exceptions already pointed out, were beyond criticism. They are truthful reproductions of what a well-trained eye will see under a good microscope when the same territory of pathological anatomy is traversed in the manner followed by the author. They must therefore possess an enduring value, whatever may be the final judgment upon the theories and opinions recorded in the explanatory text accompanying them.

The first volume of these valuable "Studies in Pathological Anatomy" comprises twenty-four royal octavo parts, illustrated with ninety-three full-page plates.

The first fasciculus appeared February, 1878, the last during May (?) 1882, the twenty-four parts being comprised in thirteen fasciculi.

The subjects and the order in which they are discussed are as follow: connective tissue, the pleura and its inflammations, the peritoneum and its inflammations, pneumonia, acute miliary tuberculosis, chronic pulmonary phthisis, acute phthisis.

It is the intention of the author to continue the publication of his studies in pathological anatomy until he has described all the lesions with which he is acquainted; but the original plan of publishing in monthly parts having been found impracticable, will be abandoned. The fasciculi will be issued of varying size and at different intervals.

In the second volume Dr. Delafield proposes to complete the description of the lesions of chronic pulmonary phthisis.

This is a work on pathology which no earnest student can afford to pass by. The great value of the first volume increasing in excellence as it does from the first fasciculus to the last, warrants the expectation that the forthcoming volume will be at least up to the high standard of the first. [E. O. S.]

Two Hard Cases. Sketches from a Physician's Portfolio. By W. W. GODDING, M.D., Washington, D. C. Boston: Houghton, Mifflin, & Co., 1882, pp. 257.

This little volume, which the publishers have made so neat that it is a credit to the art of book-making, by its obscure title, leads one to glance at its contents. The preface implies that psychological problems are to be considered, and vague hints are thrown out as to the author's intentions; not sufficient, however, to alarm the unwary. He shrewdly preludes his second hard case by hard case No. 1, and having fascinated his unsuspecting reader with his charming style and candid manner, by the recital of a sad case, the nature of which is left to the decision of the reader, he boldly launches out, under the heading: "Trial of Guiteau: Outlines for a Psychological Study," with a review of a subject which has saddened and sickened the public so long, that rest, for a time at least, was to be hoped for. In vain, however. The ghost of this great tragedy will reappear again and again. The essay before us is one of the first of a long series that will follow. The advantage of being first in the field, in this case, however, is more than offset by having to address an audience who, wearied and disgusted by a long, fatiguing play, take little interest in a recital of it after the last act is over. Yet, as when satiated with a certain article of food we may not only tolerate but actually enjoy it if it be served in a different manner, in this case, also, we find that the author has so spiced the disagreeable matter with choice language that half its bad savor has left it, and has served it so daintily that relish displaces disgust. The simple, yet elegant manner of its presentation makes it a pleasure to follow the author in his dramatic representation of this great case. It is principally a recital of the facts relating to Guiteau's life and trial; yet no opportunity is lost by the author to enforce his own opinions in favor of Guiteau's insanity. His respectful treatment of the opinion of others, however, and his evident desire not to distort facts, or to misrepresent the views of his opponents, is in such marked contrast to much that has been presented to the public that it entitles him to praise. He has maintained the true spirit of historical criticism, and has shown that it is possible to hold decided opinions without distorting them by personal animosity. On the contrary, it may be considered by some that his efforts to do justice to all, make his eulogistic treatment of the principal characters of the trial appear rather high-colored. There appears occasionally a vein of humor, not out of harmony, however, with the gravity of the subject. For example, in referring to the division of labor among the medical experts retained by the government, he states : " To Dr. Fordyce Barker, the distinguished professor of obstetrics from New York, was assigned the delivery of the definitions and limitations of the disease known as insanity."

Of the witnesses called by the defence he refers in detail to two only : namely, Drs. Kiernan and Spitzka. Respecting their testimony, he says : " The defence opened with Dr. Kiernan, a young man from the West, who was formerly connected with the hospital on Ward's Island, and now a practising physician in Chicago,—also an editor of the 'Medical Review,' and a lecturer on mental diseases at one of the colleges. It might well be thought an embarrassing position in which to place a young man, comparatively unknown, to open the medical expert testimony of this celebrated case, on which the eyes of the world were looking, though I do not know as Dr. Kiernan felt it so particularly. But after establishing his qualifications to speak as an expert on insanity, Mr. Scoville contented himself by asking him a single hypothetical question. This being answered, Mr. Scoville turned him over to cross-examination. It was apparently a pleasure to Mr. Davidge to converse with this young man. At the second question, he drew out the fact that the witness did not believe in a future state of rewards and punishments ; and though this was not, strictly speaking, a scientific refutation of the prisoner's insanity, it was, in effect, to make a Philistine of this witness to the jury, who had stated under oath that they believed in the doctrines of the Christian religion. Dr. Kiernan bore up manfully under the keen questioning of the veteran lawyer, until he drew out the further statement that five out of every twenty-five persons that one would casually meet on the street were on the road to the insane asylum, which finished this witness with the jury, for, as Mr. Davidge said, it would land two of their number there if it were true."

" The testimony of Dr. Spitzka was well delivered, and highly entertaining to the audience ; I think that really prevented its producing any lasting effect on the minds of the jury.

The doctor was in court after the issue of a compulsory process, which the counsel for the government evidently believed was done to heighten the effect of his very positive testimony for the defence. He had already criticized the district attorney's conduct of the case in unsparing terms in the public journals; he held in perfect contempt the line of experts who sat in front of the witness-box, a feeling that was apparently reciprocated. The doctor stated that he found Guiteau insane—'the marked feature of his insanity a tendency to delusion or insane opinion, and to the creation of morbid and fantastical projects, — a marked imbecility of judgment; and while I had no other evidence than the expression of his face, I should have no doubt he was also a moral imbecile, or rather a moral monstrosity.' He had decided ideas about the 'monstrosity' that he was analyzing; he had his knowledge well in hand, was quick at repartee, had an assurance that was all-sufficient; he had met most of the 'bad four' on previous occasions, and was not afraid of them, and rattled off his testimony with a volubility that was bewildering, and a positiveness of assertion that was startling to science. It was soon clear that the cross-examination was going not so much for Guiteau as Spitzka, and the result was a brilliant gladiatorial contest of some hours, without discomfiture to the witness, who left the stand with the general impression in the room that this was an 'agnostic' who did know, and what he knew he knew for certain. His estimate of Guiteau was in many respects correct. 'The congenital' character of his case, in part, is at least probable; there is some asymmetry of the head, but that of the mind is obvious; and I have never had an hour's conversation with him without being impressed with the talk as that of an insane man, though less by its 'farrago' than its leading ideas and general tone.' But the effect of the solid points in the testimony was lost in the brilliancy of the rejoinders, and I doubt if the jury got beyond thinking the doctor a very clever man, who was fully a match for Mr. Davidge."

When the testimony and opinions of Dr. Gray are considered the author enters into discussion, and expresses his opinions more freely than elsewhere. Referring to Dr. Gray as "the medical advocate and expert for the government," who "was called upon to decide whether Guiteau should be brought to trial," he quotes his definitions and opinions at length, taking issue with him on his views of insanity. He says: "I may agree with Dr. Gray in what he says so well about 'moral insanity,' and yet be willing to admit that those cases, of which the books record so many, had a real existence, while holding for myself that the disordered mind does not cease to be a unit, although the observed manifestations of its insanity may seem to be confined, in some cases, to the emotions ; in others, to the affections ; and in still others, to the intellectual powers. We cannot deny that the old masters were as keen-sighted observers as ourselves. I dislike to hear drunkenness called dipsomania, as I so often do ; but I do not therefore say that dipsomania is only drunkenness. It might improve my standing with the legal fraternity if I should pronounce kleptomania only another name for stealing ; but my personal observation convinces me that the insane have sometimes a disposition to steal, which is a direct result of their disease, and for which they are no more accountable than the puerperal maniac is for her oaths." "Heterodox I know it is, but observed facts compel me to be heterodox with Prichard and Esquirol and Ray, with Morel and Griesinger and Maudsley, and I know not how many others, in recognizing in some cases a condition inherent, born in the individual, and not a result of education,—a condition which writers have recognized as hereditary mental disorder, insane diathesis, insane temperament. But the difficulty is not in the nomenclature, but in getting scientific observers, our leaders in psychiatry, to recognize the fact as it exists." * * * "It is Gray now, but it was Ray then ; and I wondered, if that mental giant could come back from the shore where he has so lately gone to sleep, if we should not hear some such vigorous English as this : you cannot get rid of a fact by denying its existence. That is the difficulty, as I conceive, with Dr. Gray's insanity ; he simplifies our psychological studies wonderfully ; but what are we to do with those 'minds diseased' which his classification leaves out to shift for themselves ? We must still keep the bounds of insanity essentially where they were, or if we narrow them, with Dr. Gray, we must set up another kingdom of diseases and call it unsoundness of mind." The author says elsewhere, and with considerable truth, that "most of the mistakes and misunderstandings in the discussions of insanity result from an artificial nomenclature of mental diseases." He adds, however, that "it is enough to have satisfactorily established the unsoundness of a mind without being curious to label it." We think it would come nearer the truth to say that not enough effort is made to classify. A true classification—the segregation of the like—is not mere labelling. The separation of the forms of insanity into natural groups, and a recognition of the relations of these groups to each other, is required for a true classification, and this can never be accomplished as long as we are satisfied with having established "unsoundness of mind" alone.

[W. R. B.]

Epilepsy and other Chronic Convulsive Diseases: their Causes and Treatment. By W. R. Gowers, M.D., F.R.C.P., London: J. and A. Churchill, 1881, pp. 309.

By the critical study of quite a large number of cases (1450), Dr. Gowers has given us a valuable contribution to the general knowledge of epilepsy, but it must be confessed he has been able to add nothing to our knowledge that is really new, or that constitutes an advance. The etiology and semeiology of the disease are exhaustively studied statistically and analytically ; the subject of auræ being particularly well done, and the important relation of distinct auræ as indications of the seat of the morbid process (if not lesion) in a given case, is pointed out. In one half of the author's cases there was an aura, either general or limited. In giving nearly ten pages to the consideration of the "unilateral commencement" of convulsions, Dr. Gowers places himself in contradiction to his previously expressed limitation of epilepsy, for in the light of recent autopsies a large proportion of cases in which the fits begin in a unilateral manner are cases of localized coarse cerebral disease (often tumor) of the cerebral cortex. Chapter iii contains an admirable description of the spasms and general march of symptoms in an attack of epilepsy. It seems to us, however, that the author's statement that clonic convulsions (in grand-mal) are epileptic and not asphyxial is by no means substantiated. The occurrence of an asphyxial state after even a short, sometimes unrecognized (by non-experts) tonic spasm is quite certain ; and the resemblance of the phenomena of this stage to those observed in asphyxiated or suddenly anæmiated animals is wonderful. In Chapter iv, minor attacks are studied in some detail. We notice the omission from the long list of forms of petit-mal, of sudden and momentary falling to the ground with unconsciousness, a variety we have seen. Momentary and very sudden dropping of the head upon the breast we have also observed. And in our experience from a close questioning of the witnesses of attacks, it would seem that momentary general tonic spasm was more common than in Dr. Gowers' record. A fixed or statue-like state of the muscles we believe accompanies many, if not most, of the varieties of petit-mal.

In connection with minor attacks, Dr. Gowers rather too briefly treats of the mental state of epileptics, epileptic mania, interparoxysmal mental state, and chronic mental failure. In giving a statistical account of this last form of psychic disorder a most important point is omitted, viz.: the relation between epileptic dementia and the new treatment by bromides. Relatives of patients, sometimes patients themselves, raise this question, and ask if the bromide treatment will not hasten or even produce chronic failure. It would be a useful investigation to determine by means of large figures, whether epileptic dementia is more frequent than it was previous to thirty years ago.

An elaborate study of "hysteroid attacks" in patients of both sexes, leads the author to the conclusion that "the two forms of disease are not separated by any fixed and impassable symptomatic boundary." Admitting this to be true, though with all due regard for Dr. Gowers' skill as an observer, we doubt the frequency of such resemblance, we still have positive diagnostic means in a consideration of the patient's general medical history, and in trial of treatment. It has repeatedly occurred to us to reach a diagnosis by the therapeutic test in instances where the symptoms of seizures were equally epileptic and hysterical. The positive test of subsidence of attacks under the influence of bromides is open to the fallacy pointed out by Dr. Gowers on p. 180, that these drugs are often powerless against petit-mal. The negative test, however, *i. e.*, the *aggravation* of hysterical and hysteroid attacks by bromide treatment, is of much greater value.

Chapter x, on the pathology of epilepsy, is unsatisfactory, as it could hardly help being. The vague and unpathological doctrine of "discharges" is ingeniously brought forward and supported. It seems to us that the author hardly emphasizes enough, as a preliminary to this discussion, the distinction which should be rigidly maintained between the theory of the pathology of the disease and that of the mechanism of attacks. In considering the former, the insufficiency of recent microscopical observations upon the brains of epileptics is pointed out. No reference is made to Brown-Séquard's remarkable labors in the artificial production of epilepsy, though this is partly justified by the quotation as a footnote on p. 209 of Dr. J. Russell Reynolds' opinion, that artificial convulsions in rabbits and guinea-pigs are not epileptic. Now, Prof. Reynolds' excellent work on epilepsy (from which this footnote is taken) was published in 1861, before Brown-Séquard had fully developed his results in guinea-pigs. That these animals, after injuries to the cord, brain, and sciatic nerves, have truly epileptic attacks, and that some of them transmit epilepsy to their offspring, can hardly be doubted by any one who has worked with Brown-Séquard. And it occurs to us that these experimental epilepsies throw much light on the probable pathology of human cases. And in this connection we might add that a thorough discussion of reflex or peripherally produced epilepsy (in man) is a desideratum not filled by Dr. Gowers' chapter. With reference to the mechanism of the attacks themselves, it seems to us, admitting the want of sufficiency of any current theory, that the vaso-motor theory is still worthy of more consideration than Dr. Gowers accords to it ; especially if it be borne in mind that the general vaso-motor centre lies very near the "convulsive centre" of Nothnagel. The importance of dilatation of the pupil at the moment of attacks both of grand-mal and petit-mal, as corroborative of the theory of vaso-motor spasm, is overlooked. No combination of "discharges" from various parts of the brain would account for this symptom ; indeed, motor "discharges," as through the muscular nerves of the iris, would of necessity produce contraction of the pupil ; whereas we know from the study of section and stimulation of the cervical sympathetic nerve, that the movements of the iris are largely vaso-motor.

Chapter xiii, on treatment, is not as wholly satisfactory. The details of the bromide treatment are stated more fully than in any other work upon epilepsy, but not as minutely and circumstantially as the importance of the subject and the reputation of the author would demand. The various bromide eruptions are not mentioned ; only the bromide "rash" or acne ; it would appear as if Dr. Gowers had not met with the other eruptions (fully enumerated in Duhring's "Diseases of the Skin," Phila., 1882) which often resist arsenic. Bromic mania is not mentioned. The most important fault in this chapter, and one which will doubtless lead many practitioners to disastrous failure in the treatment of epilepsy, is the limit of time during which bromides should be continued after a last attack. "Hence treatment should be continued, without any reduction in dose, for six or twelve months after the last fit" . . . "A good plan, for instance, is as follows: If the attacks cease on a scruple of bromide three times a day, this should be continued for twelve months ; given twice a day, for three months more ; then half a drachm once a day substituted, and reduced three months later to a scruple, and three months later to ten grains, which may be omitted at the end of the second year."

We earnestly believe the above limitations to be fallacious and dangerous. From a tolerably large experience, and with the support of several high authorities (Voisin, Brown-Séquard, and others) we would assert that there is no safety from a return of the disease under four years. Voisin is disposed ("De l'emploi du bromure de potassium dans les maladies nerveuses," Paris, 1875, p. 65) to place the limit of treatment (with diminished doses) at ten years. For our own part, we have notes of a number of cases of epilepsy in which the patients have been over three years without attacks of any sort, yet we are not willing to publish them as "cures." In view of these statements Dr. Gowers' (and other writers') tables of results, and figures of "cures," need considerable pruning, or must even be altogether rejected.

Considered as a whole, the work of Dr. Gowers is a valuable addition to the library of the general practitioner and specialist. Many of the faults we have been obliged to point out are inseparable from the obscurity of the subject, and will doubtless exist in the monographs on this subject which will appear hereafter.

[E. C. S.]

Traité des Névroses. Par A. AXENFELD, professeur de pathologie interne à la faculté de médecine de Paris, etc., etc. Deuxième édition augmentée de 700 pages, par HENRI HUCHARD, lauréat de l' académie, etc., etc. Paris, 1882.

Against this work, first published in 1863 with 520 pages, and now, almost twenty years later, re-issued with 1,193 pages, hardly any complaint can be entered, except on account of its bulk.

Terseness of style and condensation at the expense of the elegant language which now characterizes the book would be, perhaps, after all, regretted. The additions by M. Huchard, enclosed in brackets, do credit to the author.

A well-selected and voluminous bibliography precedes each article. As an exhaustive treatise on neuroses, it necessarily includes many diseases considered by some to be organic.

Full recognition is given to the pathologists, and free discussion to the theories of both sides. Particularly is this the case in the articles on chorea and paralysis agitans. In other disorders, as angina pectoris and epilepsy, some cases of which have an unquestionable pathological anatomy, while in others, after the most careful search, we must be content with a theoretical pathological physiology, both sides are separately and fully discussed.

The three books comprising the work are: first, neuroses of sensibility (including neuralgias, migraine, vertigo, spinal irritation, angina pectoris, arthralgia, and anæsthesia); second, neuroses of motility (including convulsive facial tic, contractures, writer's cramp, dysphagia, spasm of the glottis, nervous palpitation, paralyses of reflex origin and following acute disease, and the various tremors); the third book treats of complex neuroses (including chorea, epilepsy, neurasthenia, catalepsy, and hysteria).

In the treatment of trigeminal neuralgia crystallized aconitia is advised in from .00025 to .002 and .003 doses, an amount to which we seldom, if ever, push the drug without getting profound and sometimes alarming physiological effects. Huchard advises a preliminary trial of .01–.04 doses of the alcoholic extract of aconite root. Here and elsewhere the treatment is not gone into with sufficient detail.

The surgical procedures for the permanent relief of intractable cases of trifacial neuralgia are not, it would seem, quite accurately enough described. Again, in two pages on the treatment of migraine, sixteen remedies are mentioned, but specific directions as to use are given in one case only. The Indian-hemp treatment of this disorder is simply hinted at.

The differential diagnosis between pains due to disease of the sciatic nerve and spinal cord is not explicit enough. He distinguishes pains from a diseased cord: "1st, by their seat in both inferior extremities at once, in their continuity, and in certain particular points, as the soles of the feet, where neuralgic pains rarely abide; 2d, by their form, there being dull pains, formications, numbness, and pricking rather than shooting pains; 3d, by their intensity being less than sciatic pains," etc., etc.

Both authors seemingly overlooked the pains of locomotor ataxia, which may be (1) unilateral, are generally (2) shooting, and which are usually more severe (3) than sciatic pains.

In the treatment of sciatica, pages are allotted to hydrotherapy, electricity, and parenchymatous injections, while hardly a word is devoted to the actual cautery upon which we rely so much and with reason.

Under the head of chorea, considerable prominence is given to the treatment by bromide of potassium, on which the author evidently placed much reliance; while hardly the attention which it merits is given to the arsenic treatment, so long established and proven. The administration of Fowler's solution of arsenic hypodermically is advised, but no mention is made of the dilution and filtration of the solution needed to lessen its irritant qualities.

In the treatment of epilepsy bromide of potassium is advised up to 6.–8. daily, or to the verge of mild bromism. A decided preference is given to the potassium salt.

With the exception of trivial shortcomings, some of which have been noticed, and which may even be simply differences of opinion, the book is classical, unique in its field, and worthy careful study. [R. W. A.]

A Treatise on the Science and Practice of Medicine, or the Pathology and Therapeutics of Internal Diseases. By ALONZO B. PALMER, M.D., LL.D., Professor of Pathology and Practice of Medicine, and of Clinical Medicine, in the University of Michigan; Physician to the State University Hospital, etc. Vol. ii. New York: G. P. Putnam's Sons, 27 and 29 West 23d Street, 1882.

The second volume of this work maintains throughout the eminently practical character, which was evident in the pages of the volume which we have already referred to. Practical suggestions in treatment are multiplied, until we sometimes are forced to think that their number may possibly serve to bewilder the student. The style is easy and flowing, and that the book will maintain the popularity of the author can readily be predicted.

After brief introductory remarks on physical diagnosis, under the diseases of the larynx, the author, in discussing croup and diphtheria, adopts the view of their duality. Pneumonia receives considerable attention; the various kinds spoken of by Delafield are referred to, and, ranking with the majority, pneumonic fever, or pneumonia arising from infection, is spoken of as distinct from the ordinary form, which follows the classical course and results from exposure to cold or wet.

The various theories held as to the nature of phthisis are well presented to the reader.

Diseases of the heart are briefly considered.

The chapter on kidney lesions and their symptoms is quite clearly put forth, and a division, partly English and partly German, adopted.

The author devotes more than one third of the second volume to the diseases of the brain and spinal cord, and with some twenty pages on the subject of parasites terminates his work.

To the students who have listened to his lectures during his many years of usefulness to the institution which he serves, these volumes will serve as instructive memoirs of their former teacher, and to those who are to listen, as an aid in more rapidly acquiring his views. [H. N. H.]

Nitro-glycerine as a Remedy in Angina Pectoris. By William Murrell, M.D., M.R.C.P., etc. Detroit, 1882.

As the preface says : "The object of this work is to give directions for the administration of nitro-glycerine as a remedy for angina pectoris, the principal points being illustrated by reference to cases that have been under my care. Some of these cases were published in the *Lancet*, 1879."

After a *résumé* of the mode of discovery and manufacture of the chemical, its physiological and toxic effects on frogs and cats are given. Very little time or scientific accuracy seems to have been given to this study however. The physiological effects on man are allotted twenty pages, and are pretty satisfactory.

In from two to three minutes after the ingestion of from 0.0006 to 0.003 of the drug a fulness in the head commences, which may become intense. An extreme throbbing in the head which may become general. A rushing sound in the ears has been noticed. Mental confusion going on to insensibility has occurred, and a disposition to remain quiet and to sleep is generally present. Nausea, pallor, and perspiration, some experience ; and a headache always follows, of a few minutes' to several hours' duration. In a case of epispadias, Dr. Murrell demonstrated immediate polyuria. The pulse is quickened, and, as the sphygmographic tracings introduced show, the arterial tension falls about the second minute, reaches its minimum in nine minutes, and does not entirely reappear for twenty-six minutes.

The cases introduced, twelve in number, are not all true angina, but similar. Almost all were relieved, some permanently, by the remedy.

At the close of the book the author advises the use of a one-per-cent. alcoholic solution, in doses of from 0.03 up, according to effect, every three hours, and an extra dose at the onset of the attack. Preparations in pills or chocolate tablets may be had.

[R. W. A.]

Index-Catalogue of the Library of the Surgeon-General's Office, United States Army. Vol. iii. Washington, 1882, pp. 1,020.

Americans may well be proud of the great work which is being prosecuted by the surgeon-general's office. The completion of this index-catalogue will prove a service to the medical profession throughout the entire world, and will no doubt serve to stimulate study and research. A glimpse through this third volume will serve to show the vastness of our professional literature, encouraging some students, but probably discouraging many more. Who is not, for example, appalled at the bibliography of diphtheria, occupying 29 folio pages, in double column, mostly printed in small type? The amount of clerical labor expended upon this work must be enormous, and its fidelity extraordinary, as witnessed by the rarity of errors in the spelling of various names and titles in different languages.

The present volume includes the words from "cholecyanin" to "Dzondi," distributed in 1,020 pages. The length of the article on diphtheria has been mentioned; that on Asiatic cholera occupies 157 pages—a space out of all proportion to the importance of the disease in the practice of American physicians. Chorea takes up 12 pages; climate, 6 pages; color-blindness, 4 pages; conjunctivitis, 19 pages; convulsions, 10 pages; cornea, 16 pages; croup, 23 pages; dysentery, 20 pages.

An interesting part of the volume, and one quite unique in medical catalogues, is the large list of portraits of medical men, 4,335 in number, arranged alphabetically in 21 pages. [J. W. D.]

A System of Surgery, Theoretical and Practical, in Treatises by Various Authors. Edited by T. HOLMES, A.M., etc. The American edition, from the second English edition, revised and enlarged by JOHN H. PACKARD, A.M., M.D., assisted by a large corps of eminent American surgeons, in 3 volumes 8vo, pp. 1007, pp. 1063, and pp. 1059. Philadelphia: Henry C. Lea's Son, 1881–1882.

This standard work is issued as a companion to the three-volume American revised edition of "Reynolds' System of Medicine," published by the same firm in the years 1879–80. The two publications, uniform in style, constitute fair compends of the two departments of medicine, surgery, and medicine more strictly speaking.

The system of Reynolds was revised by one man, and suffered in consequence, because, in spite of assertions to the contrary, one mind cannot be master of our recent additions to knowledge in all the departments of internal medicine.

In the surgical treatise the names of the collaborators is a pledge that in most instances the best special knowledge has been used in the revision. Without being invidious we may mention: Tumors and cancer, by Morris Longstreth; wounds and diseases of vessels, and aneurism, by Lewis A. Stimson; fractures, by the editor, Dr. Packard; hysteria, by J. S. Jewell; injuries of the head, by John A. Lidell; diseases of the urinary organs, by Edward L. Keyes; diseases of the nose and of the larynx, by J. Solis Cohen; diseases of bones, joints, and muscles, by Thomas M. Markoe; diseases of the spine, of joints, and o thopedic surgery, by E. H. Bradford; diseases of nerves, by Roberts Bartholow; gunshot wounds, by Hunter McGuire; plastic surgery, by T. G. Morton; parasites, by Joseph Leidy; surgical diagnosis, by the editor; on hospitals, by Norton Folsom. An entirely new article, of marked excellence, on diseases of the skin, by Dr. Arthur Van Harlingen, is a welcome addition. If the high reputation of authors and revisers is an evidence of the excellence of a work, then this convenient edition of "Holmes' Surgery" is all that can be wished. On the other hand, the objection may be made, that adding a few notes to articles which are somewhat out of date is a little like "putting new wine into old bottles." The reader must absorb the original and then correct his knowledge by the foot-notes and inserted paragraphs; an unfortunate process, psychologically speaking. It is probable that because of this objection many will prefer to purchase a wholly new work on surgery, even though it be the production of less celebrated pens. [J. W. D.]

A Treatise on the Physiological and Therapeutic Action of the Sulphate of Quinine. By OTIS FREDERICK MANSON, M.D., Professor of Physiology and Pathology in the Medical College of Virginia. Phila., 1882, 164 pp.

Historical remarks occupy the first eight pages. The next eleven pages are given up to the narration of some of the physiological effects of quinine on animals. Experiments, chiefly by Mêtier and Briquet, are quoted, which seem to indicate that quinine is first an excitant and then a sedative to the heart and brain, and that in the blood the fibrine was increased and the globules were relatively diminished by large doses.

The effects on man in health, occupying the next seventeen pages, embody the ideas of Magendie, Bailly, Giacomini, Guersaut, Piorry, Harrison,[1] and Wood; nothing new or original, however, appearing.

A conclusion of the study is that quinine first excites the brain and heart by exciting the stomach; that in large, repeated doses it has a calming and sedative action on the circulation and nervous system which, be the doses sufficiently large, finally paralyzes; that in large doses it produces sleep and relieves pain; and, lastly, that even in health it affects the calorifacient function, markedly.

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[1] *New Orleans Medical and Surgical Journal*, ii, 331.

As a result of forty years' observation of malarial diseases, the author advises the administration of quinine in such diseases during the hours when the febrile phenomena are naturally subsiding, which generally are during the later hours of the night. While not prohibiting the administration during the exacerbation, he says it is a very unfavorable time, on account of the difficult absorption of the salt. The stomach is very irritable, and cinchonism is more readily produced. The description of a paroxysm of remittent fever, pp. 52–58, is concise and graphic.

In yellow fever, p. 117, he considers quinine ineffectual. He thinks given early in typhoid fever it may lessen the violence of the febrile symptoms and shorten the fever.

In typhus, it is his conclusion, from the authorities referred to, that quinine does good. In the treatment of traumatic and idiopathic erysipelas it has given the author "invariable success." Almost equal faith in its power over scarlatina and pneumonia is revealed in the next few pages.

Its use in croup, delirium tremens, acute and chronic rheumatism, cholera infantum, cerebro-spinal meningitis, and surgical shock completes the therapeutical part of the book.

Notes on the "modus operandi" of the drug and the mode of administration follow. Under the latter head the oral, rectal, endemic, and hypodermic methods are enumerated. A puff for Powers and Weightman closes the book.

The great efficacy of quinine in the hands of our author is no doubt due to the engrafting of a malarial element on diverse maladies. Whether or not the presence of some palludial miasm in the author's encephalon causes the frequent repetition of the announcement of "forty years' experience with malarial fevers," we do not know. For a person who has lived so long in a malarial region, where quinine is given in all diseases, from corns to baldness, we are happy to see the author does not consider it a panacea. The fearless way in which the author handles the drug deserves all praise in these days of triturations and pellets.

[R. W. A.]

ORIGINAL OBSERVATIONS.

A CASE ILLUSTRATING THE COINCIDENCE OF DISEASES: CERVICO-BRACHIAL NEURALGIA AND ANEURISM OF THE INNOMINATE ARTERY.

By E. C. SEGUIN, M.D.

The influence of diagnosis upon therapeutics and upon prognosis has seldom been more strikingly shown in my experience than by the following case:

Mr. S. S., aged fifty-two years, consulted me June 5, 1882, for a severe neuralgic ailment of the right side of the head, neck, and arm. He related the following history: Early in the summer of 1881, he had been thrown out of a carriage upon the sidewalk, but received no evident injury. After this fall he was restless and nervous, felt badly, had more or less gastric disorder. Spent the month of August in Saratoga, but was unrelieved. About that time he first noticed pain near the right olecranon process. This pain was quite localized at first, but soon later it extended toward the shoulder; very gradually increased in extent and severity, occurring in more frequent paroxysms. Late in the autumn the shoulder region was involved; and in December pain was felt in the head, a little to the right of the vertex, and later behind the right ear. Downward the pain has extended to the hand; the fingers have never been painful or numb. The pain has been somewhat nocturnal, but never periodic. The patient has suffered extreme agony for months, pain extending from the right parietal region down the neck to the right shoulder and arm. No treatment until December, then for two months Mr. S. was under the care of a specialist for diseases of the nervous system. Has recently been at the Hot Springs of Arkansas, where, with some internal treatment and applications of hot water in bags, he was somewhat relieved. Two weeks ago was subjected to strong electrical applications which greatly aggravated his neuralgia, particularly increasing the occipito-parietal pain. Since the electrical applications, the right arm has felt big and tight (not exactly numb). After paroxysms of pain the veins of the right arm appear full. A paroxysm occurs in my office, with chief acute pain behind right ear; it is evident that the patient suffers extremely; perspires during the attack. Exertion, use of arms, or walking, causes increase of pain, or even produces a paroxysm. Fortunately no morphine habit has become established. No syphilis.

Examination: Patient presents the usual facies of prolonged suffering; is pale and thin. Seat of pain as above stated; occipito-parietal, cervical, and brachial. Right pupil a trifle larger than the left. Nerve-trunks not tender, but painful regions are hyperæsthetic in paroxysms of pain. There is no paralysis, anæsthesia, or muscular atrophy; the movements of the arm are free, except at the shoulder joint where some resistance and crepitation from false ankylosis.

The right radial pulse is very feeble, much smaller than that on the left side. The right carotid pulse is likewise much smaller than the left. The right hand is slightly swollen and tumid.

There is no sternal or pectoral deformity, but the supra-clavicular regions are both full, without yielding any unnatural pulsation. The heart is rather large, and at its base is a rough double murmur, which can be traced upward to a point of maximum intensity over the junction of the right second rib and sternum. No fulness or pulsation in supra-sternal notch. There is slight hoarseness. Many of these points were determined at a second examination.

Taking into consideration the place of beginning of the pain, its distribution, and its typically neuralgic nature on the one hand, and on the other hand, the absence of pain near the seat of intrathoracic disease, I made the double diagnosis of cervico-brachial neuralgia, and aneurism of the innominate artery. Contrary to what Mr. S.' former medical advisers had said, I did not believe that his neuralgia was a sympathetic or reflex pain dependent upon the aneurism, and hence incurable. I thought the coincidence a fortuitous one.

Acting upon this belief, I at once began treatment by cauterizing the neck with Paquelin's instrument, giving a deep injection of morphia over the brachial plexus, and ordering 4. of Thompson's solution of phosphorus (equal to about .003 of phosphorus) to be taken every three hours. In forty-eight hours very great improvement had occurred; no pain in the head since cauterization, and only two paroxysms in the shoulder and arm. Several cauterizations were made; morphia given by the mouth for a few nights; the arm was kept quiet. In about two weeks iodide of potassium was substituted for the phosphorus, and quinine also given. The neuralgia had almost ceased by the end of June; but the whole arm felt queer, heavy, and swollen (semi-painful); the radial artery was smaller, the substernal dulness more marked, and the double murmur over the innominate artery louder. In other words, while the cervico-brachial neuralgia was nearly cured, the aneurism was making progress.

Occasionally, there was slight return of cervico-brachial pain.

On August 23d, Prof. Austin Flint corroborated the diagnosis of aneurism of the innominate artery, and suggested a trial of Tufnell's rest and low-diet treatment. This the patient has decided to submit to. He has no neuralgia (none to present date, Sept. 8th); he is troubled by a severe cough, with bronchial catarrh; his right arm is puffy and bluish, and feels badly. The local physical signs in the chest are the same. He takes quinine after breakfast, and a gramme of iodide of potassium in infusion of digitalis four times a day.

I am led to publish this case because of the belief that had I looked upon the neuralgia as sympathetic and expended my therapeutic efforts upon the aneurism as the *fons et origo mali*, I should have also failed to relieve the patient.

NOTES ON A CASE OF ACUTE PHTHISIS, SHOWING THE FUTILITY OF HEROIC ANTIPYRETIC TREATMENT.

By R. W. AMIDON, M.D.

The patient, a girl of twenty-two, was admitted to the New York Hospital, in the service of Dr. Woolsey Johnson, October 12, 1878.

Without heredity or antecedents, she gave the history of having contracted a severe cold six months previously. Since that time, pain in the chest, cough with greenish expectoration, dyspnœa, anorexia, and emaciation made the diagnosis and prognosis only too clear.

A physical examination revealed signs of consolidation at both apices, with fine râles at both, and cavernous râles at the left apex.

The respiration was 26, the pulse 96, and the temperature 38.6° C. (101.6° F.).

The patient was simply put to bed, given some cough mixture, and on the following morning the pulse was 76, respiration 25, and the temperature 37.4° C. The daily examinations for the next twenty days revealed a continued fever, with an average morning and evening temperature of 38° and 39° respectively, a pulse of 85–105, and a respiration of from 20–30.

A first attempt at heroic treatment was made Oct. 4th, with the following result :

Oct. 4th.	T.	P.	Resp.
7 A. M. . . .	38.3°	100	23
11 A. M. . . .	38.9°	105	22
Cool bath . .			
Immediately after bath . . .	40°	108	34
11.40 A. M. . .	39.8°	103	32
4 P. M. . . .	39°	95	30

The bath was faithfully administered in the same way, and of the same temperature as those which, in our typhoid fever cases, produced such good results, and such sudden and lasting lowering of temperature. But in the present case, it was so disagreeable to the patient, who was not in that stupid, unresisting state common in typhoid fever, but on the contrary wide-awake to every thing, as hectics are, and, moreover, as it was productive of no good results, speedy or permanent, it was never repeated.

For about three weeks, only ordinary remedies were used. High fever, with non-periodic chills at intervals, continued. Occasionally a normal temperature was reached in the morning, but generally the record was :

	T.	P.	Resp.
A. M.	37.3°–38°	75–85	25
P. M.	39°–40.5°	105–118	35–44

Oct. 27th, another attempt was made to break the fever.

	T.	P.	Resp.
9.30 A. M. . . .	37.9°	100	23
Adm. salicylic acid, 5.20 . . .			
11 A. M. . . .	38.2°	74	27
1 P. M. . . .	38.2°	74	21
9 P. M. . . .	39.5°	108	32

This single large dose of the acid, although producing no disagreeable symptoms, had little effect on the rise of temperature, except to postpone it until later in the afternoon. Exhibition of the drug in equal amount, but in divided doses during the morning, was but little more effective.

Another interval of fifteen days passed, palliative remedies in ordinary doses being given.

Meanwhile the disease, as shown by physical signs, had made rapid advances. The patient ate little or nothing, had lost two kilos. in weight in six weeks, and was never free from chest pains and cough except when under the influence of morphine.

On Nov. 5th the temperature attained, in the afternoon, the height of 40.5°, and had stood above 39.5° since 9 A.M.

Nov. 11th.—New antipyreptic means were employed.

Nov. 11th.	T.	P.	Resp.
3 A.M. . . .	38.4°	92	24
Quinine sulphate 1.			
7 A.M. . . .	38.2°	84	24
Quinine 1.			
9 A.M. . . .	38.4°	95	24
Quinine 1.			
11 A.M. . . .	39.4°	95	28
1 P.M. . . .	39.7°	104	24
9 P.M. . . .	38.5°	90	24

Considerable headache, vertigo, tinnitus, and deafness ensued. The temperature attained the same height as on the two preceding days.

In a few succeeding days, while taking only simple remedies, the patient complained much of headache and photophobia, and was very irritable.

These symptoms passed off, and Nov. 17th, 18th, and 19th the patient was very comfortable, in spite of a temperature of 40.4° in the morning, and 40.1° in the afternoon of Nov. 19th.

Nov. 20th, another trial of quinine was decided on.

Nov. 20th.	T.	P.	Resp.
1 A.M. . . .	38.4°	102	28
Quinine .60			
2 A.M. . . .	38.3°	84	32
Quinine .60			
3 A.M. . . .	37.1°	76	28
Quinine .60			
4 A.M. . . .	37.3°	76	28
Quinine .60			
6 A.M. . . .	37.1°	79	Not recorded.
Quinine .40			
8 A.M. . . .	37.2°	76	"
Quinine .40			
2 P.M. . . .	37.1°	79	"
4 P.M. . . .	37.3°	80	"
Quinine .30			
6 P.M. . . .	37.8°	72	"
8 P.M. . . .	38°	80	"
10 P.M. . . .	38.5°	80	"
Quinine .60.			
Nov. 21st.			
1 A.M. . . .	38.4°	92	"
Quinine .60			
2 A.M. . . .	38.5°	89	"
Quinine .60			
4 A.M. . . .	38.4°	80	"
Quinine .30			
9 A.M. . . .	37.4°	85	"
Quinine .60			
10 A.M. . . .	38.1°	73	"
Quinine .60			
11 A.M. . . .	38.3°	89	"
Quinine .30			
1 P.M. . . .	38°	84	"
Quinine .60			
2 P.M. . . .	37.8°	82	"
Quinine .60			
6 P.M. . . .	38.3°	89	"
9 P.M. . . .	38°	75	"
Quinine .30			
10 P.M. . . .	38.3°	62	"
Quinine .30			
Nov. 22d.			
3 A.M. . . .	37.3°	67	"
Quinine .60			
4 A.M. . . .	37.6°	72	"
Quinine .60			

Nov. 22d.	T.	P.	Resp.
5 A.M.	38°	67	Not recorded.
Quinine .60			
9 A.M.	37.6°	80	"
Quinine .70			
10 A.M.	37.4°	74	"
Quinine .60			
3 P.M.	37.8°	75	"
Quinine .60			
4 P.M.	38°	80	"
Quinine .60			
5 P.M.	38.6°	96	"
Quinine .30			
10 P.M.	37.3°	65	"

On Nov. 23d, the patient complained so much of the headache, deafness, etc., that no quinine was administered, the amount of 16.5 grams having been taken and rétained during the three preceding days.

Nov. 24th. The headache was very severe.

Nov. 25th. The patient was very restless. She was crying and moaning with the headache all the time. An intense photophobia caused the patient to roll up her eyes to such an extent that the pupil could hardly be exposed. To-day, for the first time, noticed a hesitancy and deliberation in her speech.

Nov. 26th. Although crying a good deal, slept nearly all day under the influence of frequent small doses of chloral, the morphine which she took before not agreeing with her.

Since stopping the quinine the temperature had been gradually rising till to-day it attained the height of 40.6° at 3 P.M.

Nov. 27th. The patient to-day scowls, bores the pillow with the head, complains of pain in the back of the neck, which is very tender, and dysphagia. There is present partial paralysis of the left side of the face, and slight divergent squint when the eyes, which are still rolled up, can be seen. Vital signs: T. 40.3°; P. 132; Resp. 26. Administered during the morning iodide of potassium, 2.40.

Nov. 28th. The former symptoms continue, in addition to which the patient complained of general hyperæsthesia and diplopia. Gave iodide of potassium, 3.60 at 9 A.M., 3.60 at 2 P.M., and 6. at 7 P.M.—13.20 grams in ten hours. Vital signs: T. 40.6°; P. 127; R. 33.

Nov. 29th. A little better if any thing, but weak. Ordered milk punches and iodide of potassium, 6.50 in the morning, 7.20 at noon, and 7.20 at night. Vital signs: T. 40.7°; P. 180; R. 30.

Nov. 30th. The patient was collapsed and thought to be dying in the early morning. She rallied about 8 A.M., after the free administration of brandy by the rectum and hypodermically; marked photophobia and hyperæsthesia with intense headache continued. There were also marked divergent squint and occasional clonic spasm of the facial muscles of both sides. No iodide was given in the morning because of the vomiting, but at both one and six P.M. 7.80 were given. The maximum temperature to-day was 40.5°; P. 180; R. 25.

Dec. 1st. The patient has great tenderness in the abdomen, which is tympanitic, and pain in the ankles and knees, the legs being drawn up. There was involuntary evacuation of urine. The sacrum, which has been reddened and sensitive for a week, was now excoriated. The strabismus was less. The iodide was given morning and noon in 7.80 doses; none at night. Ordered 7.80 of the iodide three times a day. The vital signs were: T. 40°; P. 178; R. 32.

Dec. 3d. There was present to-day no headache, no facial palsy, little photophobia, and no strabismus; but the diplopia persists. The vital signs for the day were: T. 39.5°; P. 120; R. (not recorded).

The iodide was continued in 7.80 doses three times a day till Dec. 10th, when all cerebral symptoms had ceased, and decided iodism had developed.

All active treatment was now abandoned, and the pulmonary symptoms, which were in abeyance during the cerebral complication, again became prominent. The patient lingered for two months longer.

The only cerebral lesion found on autopsy was a thickening of the pia-mater at the base of the brain.

This case shows the impotency of our so-called antipyretics in combating the pyrexia of phthisis.

In rheumatism, salicylic acid, and in malarial fever, quinine lowers the temperature, because they neutralize the poison or destroy the germ on which the pyrexia depends.

In acute phthisis, however, besides a doubtful septic factor, we have a local destructive inflammatory process, which, unlike many of our other inflammations, shows little or no tendency to self-limitation, spontaneous or induced cure. When, therefore, the diagnosis and prognosis are clear, in our present ignorance, antipyretics hold out no hope, and the expediency of their use at the expense of the personal comfort of the patient is doubtful. One or two trials of them, in the hope that an error in diagnosis has been made, or that good in some way may accrue, is justifiable, nay, perhaps wise. Their persistent use, however, should be condemned.

A study of the above case emphasizes some of the physiological and therapeutical effects of the means employed. Some of them we will enumerate.

On the day the cold bath was given 1302 cc. of urine were secreted, against 930 cc. the day before and 558 cc. the day after. The bath was followed, immediately, by a rise in the temperature (not uncommon), pulse and respiration as usual; and, more remotely, by a fall of all three vital signs. The day on which a single large dose of salicylic acid was given was also marked by polyuria, and the usual mild cerebral symptoms. The antipyretic effect of the drug was only manifested by a postponement of the afternoon rise of temperature, pulse, and respiration until 9 P. M.

In reviewing the effects of quinine on the patient, a more interesting and perplexing field is opened. The first time when 3. of quinine was given in six hours there followed the usual headache, vertigo, tinnitus, and deafness, all of which were transitory. There ensued a few days of inexplicable headache, photophobia, and irritability, which also vanished, but were admonitions of what was coming.

November 20th, 21st, and 22d the patient took 16.5 grams of quinine. During these days, and on the 23d, the common sensory physiological effects were very marked. The febrile temperature and pulse also yielded in a very marked way (see table). On the 24th the headache became intolerable; and later, undoubted indications of a cerebral lesion presented themselves: headache, pain, and tenderness in the back of the neck, general hyperæsthesia, partial opisthotonos, dysphagia, photophobia, strabismus, diplopia, facial palsy, hesitancy and deliberation in the speech, the formation of a bed-sore, and, finally, complete disappearance of the cerebral symptoms under large doses of the iodide of potash.

Most probably the pathological condition in this instance was a meningitis. Then the question arises whether, in a patient subjected to conditions like ours, the meningitis was tubercular, or that rare and much tabooed form which many authorities claim can come from large doses of quinine.

We are inclined to the latter view for the following reasons:

Tubercular meningitis, if *ever* cured, would doubtless leave behind some local lesions in the meninges, while in this case they were diligently sought for and not found.

No history of syphilis could be proven. Large amounts of quinine, however, had been taken, and doubtless caused the diffuse, basal meningitis, whose only trace on *post-mortem* was a diffuse but decided opacity of the basal pia mater.

HYSTERICAL CONVULSIONS AND HEMI-ANÆSTHESIA IN AN ADULT MALE: CURE BY METALLO-THERAPY (GOLD).

By E. C. SEGUIN, M.D.

Examples of hysterical convulsions in the male sex are rather frequent in youth and boyhood, but after twenty they become so rare as to be worthy of record. Still more unusual is it (in this country at least) for hemi-anæsthesia to follow the succession of convulsions. For these reasons, and because the case presents points of interest as regards diagnosis and therapeutics, I desire to place it before the readers of the ARCHIVES.

James A., 21 years old, single, and a laborer by occupation, was brought to the Manhattan Eye and Ear Hospital by Dr. Smith of Newtown, Ct., for the diagnosis and treatment of an alarming set of nervous symptoms; briefly summed up as convulsions, extreme staggering, left-sided hemi-anæsthesia.

History.—A year ago the patient fell from the upper platform of a freight car, a distance of at least ten feet, striking the ground upon the back of his head. He thinks that he was unconscious for a few moments, but did not vomit. Remained well after this fall until some six weeks ago. Denies sexual excesses or irregularities. At that time, some six weeks ago, he had an ill-defined illness—apparently a severe "cold," characterized chiefly by pains all over his body, in the muscles mostly. Thinks that he had no fever (locality is malarious, however), and is positive that he had no articular swelling or sore throat. The account of the order of appearance of the nervous symptoms is obscure, as Dr. Smith did not see patient until two weeks ago. Then had already had several "fits," apparently of an epileptic nature; he was not paralyzed, but exhibited complete insensibility to pricking on the left side of his head, face, tongue, and body. He also staggered somewhat. He complained of headache, near the vertex, and over the right parietal region. Convulsions occurred every night; and one night about ten days ago there were several, which were witnessed by Dr. Smith. In these attacks the patient was stiff; eyes closed, showing, when the lids were raised, normal pupils;

the respiration was slow and gasping; the spasm was only tonic, and lasted, quite certainly, not less than three minutes. There was no frothing of the mouth, or subsequent drowsiness. The patient claimed not to know any thing of these seizures. Attacks occurred yesterday. A friend of the patient describes attacks lasting an hour and a half. The staggering gradually increased during the fortnight of observation; clear (colorless?) urine was often voided; no globus or emotional seizures. Has seemed rather obtuse or stupid. Much bromide of potassium has been administered; at first he had forty grains (2.75 grammes) every four hours, and later every two hours, and less often. Altogether, has taken about 45 grammes (℥ jss) in ten days.

Examination.—Patient is an average, dull-looking Irishman, generally pale, and with the neurotic white circle about his mouth strongly marked. Comes into the room supported by two persons; staggers preposterously; when not supported plunges off to one side or the other. No paralysis; sees and hears well (to simple tests). Pupils normal. Left side of body, face, and tongue presents complete analgesia. Ends of fingers are a little sensitive to *deep* pricking (only in last two or three days). The various modes of sensibility and the special senses were not critically studied, because we purposed doing this on another day. To watch-test and to ordinary objects there was no deafness or blindness of the left ear and eye. An interesting experiment was made upon the patient as regards his equilibrium. I placed him in the middle of the room, loosened his friend's hold of his arm, and told him to look up at the ceiling and try to see certain fine marks upon it. Thinking that I was testing his eyesight, he strongly directed his attention that way and stood *perfectly well*, without a trace of his staggering; which, however, returned the moment that the test was over and he was told to stand alone—*that* he could not do (while thinking of it).

Without saying any thing to the patient or to the physicians and students standing by, I applied two twenty-dollar gold pieces to the patient's left hand, and afterward to his forearm, cheek, and tongue. I most positively said or did nothing which could *suggest* any thing to him. He could not tell whether I meant this as a continuation of the examination, or as a remedial measure; he looked and spoke as if he thought I was amusing myself in applying the gold.

In a few moments, one to three minutes, sensibility returned in each spot where the metal had been laid; completely so in the tongue, and partially in the cheek, forearm, and hand. The patient was amazed. I ordered a capsule containing .30 of citrate of iron and quinine and .01 of extract of nux vomica to be taken four times a day.

The next day, September 21st, the patient was examined by my colleague, Dr. W. R. Birdsall, who found him nearly free from staggering; pricking with a needle was felt a little less than normally on the left face, quite normally on the left arm and hand. On the left leg (not yet treated) pricking was somewhat felt, but simple touch was not perceived. A belt of gold plates was applied round about the calf for ten minutes, when sensibility was found to be restored, not simply where the plates had been laid, but throughout the extremity. On neither day was any phenomenon of "transfer" observed.

On Sept. 23d patient, claiming to be perfectly well, left the hospital contrary to my request.

The diagnosis of the case presented but slight difficulties, in spite of its extreme rarity. The staggering was evidently overdone, or at least greater than in any organic or functional cerebral disease known to me; and it was made to cease by diverting the patient's attention in an interesting manner. The convulsions were too long to be any thing but hysterical, and the state of the pupils indicated the non-epileptic nature of the seizures. The continuation of the symptoms—nay, their aggravation—under severe bromide treatment was in accordance with my own experience in hysterical cases.

The brilliant success of metallo-therapy in this case is interesting and very puzzling. I believe that every physician present when the gold was first applied, will agree with my statement that there was no sort of *suggestion* made to the patient—nothing was said until after the patient himself looked up in amazement at the restoration of sensibility to his hand and tongue.

It might be added that in the last two years I have had several successful cases of metallo-therapy in my practice—all of them reactions to gold. One of the most striking was that of a girl aged about sixteen, showing decided chloro-anæmia, but free from hysterical symptoms except analgesia of the whole left upper extremity, and the neurotic white circle about the mouth. In this case an elongated oval area on the extensor surface of the forearm remained analgesic in spite of several short applications of the gold, but yielded to their continued contact for twenty-four hours.

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ARCHIVES OF MEDICINE.

Original Articles.

A CASE OF MYXŒDEMA.

By ELIZABETH M. CUSHIER, M.D.,

PHYSICIAN TO N. Y. INFIRMARY FOR WOMEN AND CHILDREN.

Mrs. S., æt. fifty-seven. Born in England, but has lived in this country since infancy. Has had twelve children, her last pregnancy being eighteen years since. Health had been good until she received a severe shock during N. Y. riots in '61. She was at that time living in a part of the city where there was a large Irish population, and her husband being known as a Republican and Abolitionist, his life was threatened and their property destroyed. From this time her health began to fail. She lost strength and suffered from various anomalous ailments. Within the next few years—a precise date could not be obtained—her friends began to notice a change in her face, which became gradually swollen. She also grew inactive, from having been of an energetic habit. This inactivity she attributed to a difficulty in walking. She felt it a burden to walk, and had a sensation when attempting to do so of being drawn forward. Her appetite remained good, and with the exception of what appeared to be a slight deafness, her faculties remained unimpaired. I saw the patient for the first time in the winter of '77. Was called on account of œdema of feet and legs, and various new and distressing symptoms. I found her sitting up in a chair with her head hanging forward. Her face presented a strange appearance : it was large, especially about the cheeks ; the nose was flattened, making distance between eyes appear greater, and it was also thickened ; the curve of the nostrils was lost ; the lips were thick, and the lines about the mouth were obliterated ; the eyelids were puffy and bagging, and the eyes, owing to the thickness of lids, seemed small and narrow. The general appearance of the face suggested the œdema of Bright's disease, although there were differences which became later more marked. There was at this time œdema of lower extremities, and to a slight extent also of hands. Bright's disease was, therefore, suspected, and an examination was made of the urine. The utmost care, however, failed to detect the slightest evidence of renal disease. The urine was absolutely normal. An examination of other organs, which was carefully repeated in the hope of finding some cause for the existing condition, gave negative results; with the exception of a slight increase in the area of the heart's dulness, nothing was found.

The cause of this œdema remaining unexplained, the patient was treated symptomatically, and under the use of diuretics, with strychnine and iron, the swelling gradually diminished, and finally disappeared, with the exception of that of the face, which remained in the same condition. There was but little change in the general symptoms of the patient, and she remained comparatively comfortable until the summer of 1879, when the œdema of lower extremities returned, and she had an acute attack of inflammation of skin of posterior aspect of legs. The spots were circumscribed and vesicular, and they rapidly ulcerated, and as rapidly healed, under treatment which consisted mainly of rest, bandaging, and astringent lotions. From this time—with the exception of an intercurrent attack of hemorrhoids, which were operated upon and very readily healed, nothing of special note occurred until a short time before patient's death. During this time the difficulty in walking and supporting herself in a sitting posture increased, and she remained for the most part in bed. The inability to rise or walk was peculiar. The patient would wait for some seconds after the impulse had apparently arisen, as if preparing herself for the effort. She would then very slowly rise, supporting herself by her hands while doing so; her head would remain bent strongly forward until she had risen, then by an effort she would slowly lift the head and stand erect, with the head, however, always inclining somewhat forward. When once upon her feet, and started, patient could walk, and there was no peculiarity about her gait other than its slowness and clumsiness.

There was no apparent diminution of general sensibility in extremities, at least none that could be detected without the use of the æsthesiometer, and the œdema quite disappeared only to return a few weeks previous to her death. During these years the skin had become very dry and harsh, and the face still more enlarged, and as the increase in size did not involve the forehead, the comparative narrowness here, together with absence of expression, gave the countenance a strange and stupid aspect. The cheeks were firm and waxy to the touch, and rosy pink in color forming a marked contrast to the transparent whiteness of skin around mouth and eyes. The lips had entirely lost their mobility, so that when patient spoke it was as if through a mask.

The case remained an enigma to me until about fourteen months before her death. At about this time my attention was attracted by an article by Dr. Clifford Mercer, of Syracuse, on "Myxœdema."[1] The paper in question was read before the Syracuse Medical Society, and published later in the *Medical Record.* Dr. Mercer had seen, through the kindness of Dr. Ord, of London, some cases of this disease—then entirely new to him,—and his description of them, and later Ord's account,[2] left no room for doubt as to the nature of the trouble in my patient's case, and a careful examination for further details only corroborated the diagnosis. There were the same semi-transparent skin ; the bagging of eyelids—hanging, indeed, in large folds,—the even and persistent flush on the cheek, terminating by a sharp line at lower border of orbit, and forming a marked contrast with white circular area surrounding eyes ; the flattening of nose, and increase of width between eyes ; the placid face, with thick expressionless lips of a violet tint ; thickening of tongue, and fat bolsters in superior clavicular region ; slowness of comprehension, and slowness of response ; labored articulation, and monotonous voice. All were there, making a most complete picture. Added to this were the difficulty in rising and walking, without paralysis, and also the hanging of head upon chest, above mentioned, and attributed by Ord to difficulty in co-ordination and to incapacity of will to maintain muscular action. The patient was, as in Ord's cases, well nourished. The mental processes were correctly performed—but slowly, and with an effort,—and finally there was a subnormal temperature and slow pulse. Having decided upon the nature of the case, I asked Dr. Putnam Jacobi to see the patient with me. The doctor entirely agreed with me in the diagnosis. The subsequent history of the case is briefly told.

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[1] *Medical Record*, April 16, 1881.

[2] Tr. Clin. Soc., London, 1880, xiii.

The symptoms mentioned became more marked, and the patient was confined constantly to her bed. Her appetite remained good, but there was obstinate constipation. There were occasional attacks of œdema of lower extremities, which, however, subsided, and only became permanent a few weeks before patient's death. The peculiar condition of the skin characteristic of the myxœdema, was limited to the face and neck ; elsewhere there was no permanent swelling, and none that would not—like the true œdema—pit on pressure. The skin, however, was everywhere—excepting the face—dry, harsh, and scaly. The heart's area became markedly increased, and percussion during last few months of life gave abdominal dulness, which changed with change of position of patient, and gave rise to supposition of ascitis. The abdomen also greatly increased in size. About three weeks previous to death, patient had an acute attack of facial dermatitis. It began on the left cheek and extended across nose to opposite cheek, and later to both ears. The swelling was very great, and there were vesiculation of the skin and considerable pain. A diagnosis in regard to the character of the inflammation was quite difficult, as the general appearance resembled in many of its characters an erysipelatous inflammation. The almost complete absence of fever and other constitutional symptoms determined the exclusion of erysipelas, and the rapid course and termination of the disease proved the correctness of the diagnosis. A few days previous to patient's death there were an acute swelling and increase of temperature of left upper arm, with pain about elbow joint, and also a small phlegmon on outer side of right breast. An interesting point in this connection is that the patient could not locate the pain, but constantly referred it to her right arm. This was the only evidence of any disturbance of sensation. The patient's strength from this time gradually failed, and she died on the 12th of June, 1882.

An autopsy was held fourteen hours after death with the following results :

Rigor mortis well marked. Body moderately fat. Abdomen quite prominent. Slight œdema of lower extremities. Face large and waxen. Eyelids transparent and bagging. Skin with exception of face rough and scaly.

The spinal cord was first removed. Upon cutting through integument of cervical and upper dorsal regions it was found to be very thick, and the subcutaneous connective tissue infiltrated with transparent material, giving it the appearance of the umbilical cord. This condition was not found elsewhere in any part of the skin which was divided, but it probably existed in the face. Nothing abnormal was found macroscopically in the spinal cord. An attempt was made to examine the brain, but owing to an excessive thickening and hardening of the bones of the skull, and the unfavorable conditions under which the autopsy was made, the patient being in an ice-box, the attempt was reluctantly but necessarily abandoned. Upon opening the abdomen the walls were found to be very fat, and it was noted with surprise that no fluid was present in the cavity. The omentum and mesentery were loaded with fat, and it was to this that the dulness on percussion was due. The laxity of the abdominal walls, and the weight of the fatty intestines favored the descent of the latter toward whichever side upon which patient was placed, and gave rise to the absolute dulness on percussion, and to the change of the same on changing the position of patient. The thyroid gland was somewhat smaller than normal, especially the left lobe. The latter had undergone cystic degeneration. The right lobe was of unusually firm texture. The pericardial sac contained a slight excess of fluid. The heart was enormously increased in size, both ventricles were hypertrophied, and the right also somewhat dilated. The tissue was pale and firm. The valves were normal. There was beginning atheroma of aorta. The lungs were quite normal, and there was no effusion in the pleural cavities. The spleen was normal. Liver was normal in size, but tissue was unusually firm. The kidneys were slightly enlarged, and there was moderate atrophy of cortical portion.

The following tissues were subjected to microscopic examination, and with accompanying results: Skin and muscle from dorsal and anterior cervical regions, portions of tongue, kidney, spinal cord, and thyroid gland. The liver was not examined, owing to the specimen having been mislaid.

Microscopic Examination.

The subcutaneous connective tissue was found greatly increased, and there was atrophy, and in part complete absence, of the hair follicles, and sebaceous and perspiratory glands. The individual fibrils of connective tissue appeared swollen, and there were scattered here and there groups of small round cells. The nuclei of connective tissue were distinct and large. In many places the tissue presented a glistening whiteness, and there were also present small spherical bodies which had a highly refractive power. Most of these were entirely homogeneous, but others contained finely granular matter.

The muscular fibres of heart were markedly atrophied, in many places being diminished to one third their original size. The striæ were almost obliterated, and there was a diminution in number of nuclei. There was an increase of the interfibrillar connective tissue, making wide spaces between the fibres, and these spaces were filled with small glistening spherical masses.

The kidneys presented, both in the medullary and cortical portions, atrophy of the tubes and Malpighian bodies, with an increase of the interstitial tissue. In isolated places there was complete disappearance of the tubes, and a substitution of swollen connective-tissue fibres. Here, also, were found the same transparent, white, highly refractive masses noticed in sections from heart and subcutaneous tissue. The epithelial elements of kidney were granular. In the thyroid gland the walls of alveoli were thickened, and their contents had either entirely disappeared or were replaced by a homogeneous substance. The smaller blood-vessels were mostly obliterated by the excessive thickening of their coats, caused by infiltration of hyaline substance between the fibres forming them.

There was also an increase of connective tissue, presenting the same swollen appearance, in the tongue. In places there were single muscular fibres widely separated from the remaining bundle by this interfibrillar substance; also, large irregular-shaped spaces filled with a hyaline substance.

In the spinal cord changes were found in the blood-vessels and in the nerve cells. In many of the sections the small vessels had undergone changes in their walls, resulting in a thickening and infiltration of the same, with a transparent homogeneous substance. In the lower third of the lumbar cord, in region of anterior horn, was a mass composed of dilated blood-vessels and granular pigment. In other portions of lumbar region, also in columns of Clark and in lower cervical region, slight hemorrhages had taken place, in some places the pigmentary remains of which could alone be seen. There was marked dilatation of blood-vessels throughout most of sections.

In the sections from lumbar cord, in both anterior and posterior horns, there were here and there cells which had undergone complete "yellow degeneration." There were also atrophy of the cells, and absence, or diminution in the number, of their processes. In posterior columns outside of horns, there were small spaces filled with a transparent, glistening material. In upper portion of lumbar cord the atrophy of cells was less marked, but many of them were changed in character. The central canal was obliterated.

In cervical cord, lower segment, the cells in anterior horns were more numerous than in the sections below, and the atrophy of cells in posterior horns less marked. There were also in the white columns numerous glistening spaces, and on outside of the columns, small round glistening bodies. In upper segment of cervical cord the changes were still less marked, but the cells were still diminished in number, and the atrophy of nerve tubes gave mottled appearance to sections. There was here also partial obliteration of central canal, Throughout dorsal region changes similar to those above were noticed.

I am indebted for the above results of examination of spinal cord to my friend Dr. Putnan Jacobi, and also to the kindness of Dr. Birdsall.

Since 1873, when Dr. Gull read before the Clinical Society of London, a paper entitled "A Cretinoid Condition Supervening in Women in Adult Life," up to the present time, about fifty cases of the disease in question, now generally known as myxœdema, have been reported. The name given to it by Dr. Gull was suggested by several points of resemblance in the physical character and mental symptoms of the disease, to cretinism, and it was not until six years later that any explanation of its pathology was offered. At this time Dr. Ord, of London, published a report of three cases,[1] in one of which there had been a *post-mortem*, and to him is due the interesting discovery, that the œdema was the result of an infiltration of the connective tissue with a substance containing an enormous amount of mucin.

While these investigations were being pursued in England, another observer, Dr. Morvan, in Basse Bretagne, had noted a peculiar affection occurring in women, and of which he could find no description. In 1875, he wrote to Charcot, giving an account of his cases—the description of which leaves no doubt as to their nature,—and asked for some light upon the subject which was puzzling him. After some little time had elapsed, he received word from Charcot, that neither from his own experience nor from the careful study of the most recent literature could he find any thing which would correspond to the condition in question, and he concluded by advising Morvan to publish an account of his cases. This Morvan deferred, while he continued to observe and record his cases, until in 1881 his attention was directed to an article by Merklen, in the May number of the *Gaz. hebdomadaire*, in which a case of myxœdema was described. Morvan at once recognized the disease as being identical with that which had for so long been an enigma to him, and in the following August he published a paper in the same journal, giving a detailed account of fifteen cases which had come under his observation in Basse Bretagne.[2] Among these cases (which occurred, as in the experience of other observers, mostly in middle-aged women,) were three under the age of twenty-five years, and one case occurred in a man. With one exception, the subjects of the disease were peasants. In the meantime, cases had been reported by Charcot[1] in Paris, Duckworth,[2] Lloyd,[3] and Inglis,[4] in England, and Clouston[5] in Scotland; while in this country one case had also been presented by Dr. Hammond.[6] Since that time still other cases have been brought into notice, until, so far as can be ascertained, there are now, including the one above reported, in all, fifty cases on record. It is singular that no mention of the disease—at least by any recognizable name—is found in German medical literature. A careful search in the different German journals has been absolutely without results.

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[1] Trans. Clin. Society, London, 1878, lxi, 57–78.

[2] *Gaz. hebd.*, Paris, August, 1881.

In all the cases reported the general aspect of the patient is remarkably similar, especially in regard to the appearance of the face, scaly condition of skin, absence of hair, and suppression of perspiratory secretion. There is also a universal agreement in the history of the cases in regard to slowness of speech, muscular weakness, and slowness of all muscular acts. The slowness of mental processes is almost invariably admitted, but in regard to mental disturbance other than this, there is great diversity in the statements of the different cases. Doctors Gull and Ord lay especial stress upon the fact, that all the mental acts are generally accurately performed but with unusual slowness. Dr. Morvan also in his fifteen cases reports the intellect intact with the exception of two cases. In both of these there were hallucinations, and in one of them the intellect was enfeebled. The following table will give, as far as could be ascertained, the symptoms pertaining to the nervous systems in the various cases which have been reported.

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[1] *Progrès Méd.*, Paris, 1880, Nos. 30–31.
Gaz. d' hop., Paris, 1881, l-in, 73–75.

[2] *Lond. Lancet*, 1879, ii, 577.
Med. Press and Circ., London, 1879, n. s., xxviii.

[3] *Lancet*, London, 1881, i, 138.

[4] *Lancet*, London, 1880, ii, 496.

[5] *Edinburg Med. Journal*, 1880–81.

[6] *Neurological Contrib.*, vol. i, No. 3.

	CASES.	SEX.	AGE.	SPEECH.	MUSC. SYMPT.	CEREBRAL SYMPT.
Gull	5	Female	45	Guttural and labored	Musc. languor	Unimpaired intellect. Mind indifferent.
		"	40	"	Slow movements	Mind placid and lazy. More irritable.
Ord	6	"	54	Slow	Loss of power	Strange taste. Weak-headed. Languor. Slow perception.
		"	36	"	Slow action	Slowness of thought. Ideas clear.
		"	40	"	Slow movements	Slowness of thought.
		"	45	"	" "	" "
		"	60	"	" "	" "
		"	52	"	Staggering gait	Memory defective.
Morvan	15	Female	55	Slow	Enfeeblement	Halluc. and enfeebled intellect.
		"	27	"	Gen. paresis	Intellect intact.
		"	44	"	Slow movements	Hallucinations.
		"	48	"	Enfeeblement	Has observed no diminution of intelligence in his cases.
		"	40	"	Gen. paresis	
		"	50	"	" "	
		"	22	"	" "	
		"	46	"	" "	
		"	40	"	" "	
		"	40	"		
		"	46	"	Enfeeblement	
	3	"	43	"	"	Lethargy
		"	39	"	"	
		"	67	"	"	
		Male	50	"	"	Intelligence intact.
Fournier	1	Female	55	"	"	Hallucinations in dreams, but intellect intact.
Duckworth	3	"	47	"	Slow movements	Dull and morose.
		"	46	"	" "	
		"	34	"	" "	Aberration.
Charcot	3	Male	57	"		Apathy.
		Boy	19	"		
Lloyd	2	Female	40	Thick	Slow movements	Delusions.
		"	65			
Inglis	2	Male		Slow	Slow movem'nts Dimin. farad. react.	Mind enfeebled.
		Female	39			Delusions. Silly. Childish.
Thaon	1	"	39	Slow	Movements slow	Frightful dreams. Slow thinking.
Mahomed	1	"	30	"		
Lattey	1	"	57	"		Dull and heavy.
Blaise	1	"	34	"		Halluc. of taste, smell, hearing, sight. Slowness of thought. Apparitions and frightful dreams during sleep.

	CASES.	SEX.	AGE.	SPEECH.	MUSC. SYMPT.	CEREBRAL SYMPT.
Hopkins	1	Female	49	Slow and thick	Slow movem'nts Sometimes fell Dimin. elect. react.	Memory impaired. Temper milder. Deafness. Taste and smell impaired. Sight dimmed by mists and showers of specks.
Gowans	1	"	60	Slow	Staggering gait	Slowness of thought. Mental indifference. Memory defective. Irritable and depressed at times.
Hammond	1	Female	41	Slow and sluggish	Staggering gait Dim. of cutaneous sensibility	Diminution of sense of hearing. Abolition of sense of taste and smell. Hallucinations. Failure of memory, and marked weakening of intellect.
Cushier		Female	50	Slow and labored	Loss of musc. power. Walked with effort	Deafness. Slowness in responding when spoken to; intellect otherwise intact.

In addition to above, other cases have been reported by Simon Carafy, and Fenn, but precise statements in reference to the nervous symptoms were not obtained.

The various opinions in regard to the essential nature of the disease in question are based in part upon the theory that the main anatomical lesion, *i. e.*, the mucoid infiltration, is the point of departure for the nervous symptoms, and in part upon the theory of a profound general nutritive disturbance, of which the connective-tissue change is but the manifestation.

Ord, who was the first to recognize the important anatomical change which had taken place in the connective tissue, points to the analogy existing between this general condition and that of the embryo, where the elements of connective tissue, especially those containing mucin, are predominant, and speaks of it as a reversion or degeneration toward the embryonic state. He then argues that such a condition of the skin, by interfering with the natural exposure of nerve-ends to stimuli, appears in itself an explanation of most, if not all, of the characteristic nervous phenomena above related. And as one most essential condition of health of the central nervous system is, that it receive its due amount of stimulation through skin, and receive it regularly, any such padding of peripheric nerves would result in the perception becoming slower than usual, and thus the central nervous system losing its natural stimulus, a state of intellectual lethargy and slowness in co-ordination would be the result. In his first two autopsies Ord did not find any corresponding changes in the nerve tissue. Later he admits these changes, but regards their effects as secondary to the constant, extensive, and progressive interference with function of peripheric nerves.

Fournier,[1] after analyzing the symptoms in the recorded cases, thinks the mental manifestations null in comparison with the muscular paralysis, which is present in every case observed, the importance of which is as marked as the œdema itself, and remarks that the paralysis affects all the muscles, the striated as well as those of vegetative life, and as a result we see fatigue in walking, defect in equilibrium of body, difficulty and slowness of speech, dysphagia, constipation, slowness and feebleness of heart's action and of arterial pulsation; and he concludes that the pathological process is evidently an intimate alteration of the nutrition of the cutaneous envelope and subjacent cellular tissue; and as the histological perversion is general, it is necessary to seek its cause in the acts which regulate the nutritive movements in the affected parts.

According to Morvan,[2] in whose detailed account of fifteen cases there is a marked predominance of muscular paresis over the mental symptoms, the motor nerves are paralyzed, and myxœdema is a neurosis of the nervous system, affecting the system only in the parts which pertain to the motor nerves,—as well those of animal as those of organic life,—and leaving unimpaired all those pertaining to sensation and the mental faculties.

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[1] *Gaz. hebd. de méd.*, Paris, 1882, 2 s., xix.

[2] *Loc. cit.*

Hadden,[1] after noting the diminution of temperature and diminution of urea observed in all cases where these conditions were sought, sees in some disturbance of the vaso-motor system an explanation of most of the symptoms of myxœdema, that is, diminished tissue-waste, diminution of heat, together with bodily and mental sluggishness, and asks in this connection: "If vaso-motor paralysis be the cause of the enlarged thyroid in exoph-thalmic goitre, may not vaso-motor spasm be invoked on behalf of the opposite condition of this gland in myxœdema?" He mentions, in this connection, a case of Dr. Ord's, in which exophthalmus precedes the onset of the disease, and says that a certain degree of protrusion of the eyeballs has been observed more than once. Dr. Hadden then offers the following explanation of the solid œdema: "Angiospasm does not affect the blood vascular system alone, but involves also the lymphatic. Under such conditions, there would be a generalized lymphatic obstruction, and an inability to remove the products from the lymphatic spaces," and adds, that "it is conceivable that these accumulated products should, in the course of time, undergo changes resulting in the formation of mucin."

The most recent paper on the subject is one by Henrot, which was read before the recent meeting of the "Association française pour l' avancement des sciences." Henrot gives a case in which a most careful *post-mortem* was made, and the following are his conclusions: "The disease is characterized by a generalized mucoid œdema of the subcutaneous cellular, and submucous tissues; by a mucoid degeneration of the upper and lower extremities, tongue, lymphatic glands, liver, spleen, great sympathetics, pituitary body, and pineal gland; by atrophy of the muscular tissue of the heart, and blood-vessels, and of the voluntary muscular system, also those parts of the nervous system which preside over sensation and intelligence; by a profound anæmia giving to the skin the appearance of wax without the existence of leucocythemia. In a word, the vegetative centres take an absolute preponderance over the centres of animal life."

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[1] *Brain*, vol. v, No. 2.

Henrot places the lesion which produces this general mucoid degeneration in "l' appariel cinerio hypophysaire, which is formed at the same time by a vascular gland and the anastomosis of the two trunks of the great sympathetic." He then draws attention to the study of Owen, according to whom this body is markedly developed in the embryonic state, and also in animals who, like the fish, have normally a large amount of mucoid matter, and concludes: "The disease known as myxœdema, or cachexie pachydermique, seems essentially constituted of a return of the subcutaneous, submucous, and interstitial connective tissue, to the embryonic state, under the influence of hypertrophy of the vegetative centres, and particularly of the sympathetic ganglia and the vasculo-sanguineous glands, which are especially connected with them."

In regard to the light which has so far been thrown upon the nature of the disease in question, Ord's discovery alone, based as it is upon existing anatomo-pathological conditions, is of any scientific value. The theories in regard to its essential cause are unsupported by facts, and are in part hypotheses having no known physiological data from which to infer the presumed pathological change. Until, therefore, further investigation has brought more precise knowledge from which to draw conclusions, the disease in question can only be described as a nutritive disturbance resulting in the presence in the connective tissue of a substance common in embryonic tissue, but not existing normally, excepting in very small amounts, in adult life, at which time the connecting substance of connective tissue is formed largely of collagen. As these two substances, collagen and mucin, are, it is presumed, the result of decomposition of the proteids, it would seem that the process in myxœdema is one in which the nutritive material intended for the repair of the connective substance, is converted into a lower form of tissue, one which is a reversion to an embryonic condition, whether or not at the expense of the collagen has not, that I am aware, been ascertained. The association, as in the above case, of colloid with the mucin is not surprising when the chemico-physiological relation of the two substances is taken into consideration.

The points of interest in the case recorded above are, the limitation of the mucoid œdema of the skin to the face and neck, while the muscular paresis of the lower extremities, mentioned as a prominent symptom of the disease, was very marked. At the same time, the sections from spinal cord gave evidence of changes which must certainly have been connected with this loss of muscular power.

Whether in these cases the paresis is due to a primary lesion in the spinal cord, some form of myelitis to which the hyperæmia and degeneration of cells would point, or whether the essential cause is a trophic neurosis, of which the changes in the cord are but one manifestation, upon which, indeed, they depend, are questions which at present cannot be answered ; but the identity of the changes in the blood-vessels, in the cord, and in those of the other tissues, and the presence in its connective tissue of the same abnormal products which are found elsewhere in this tissue, would point to the latter as more probable, and suggest for the disease in question a relationship to those other troph-neuroses—progressive muscular atrophy and pseudo-muscular hypertrophy,—in which the main anatomical lesion, at least in the early stages of the disease, is found in the connective tissue.

In the case reported above, to whatever may have been due the changes found in the spinal cord, they were certainly sufficient to account for the muscular paresis, leaving no need for evoking a peripheric cause.

POSSIBLE CONTAGION OF PHTHISIS.

By E. G. JANEWAY, M.D.,

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AS the possible origin of tuberculosis and phthisis by contagion has become the subject of experiment and study on the part of members of the medical profession, and of increasing importance to the public, I have thought that the following possible instances possessed sufficient value to warrant their publication.

The first is one of exceptional interest, as it shows that dogs in contact with a man suffering from phthisis became affected with a pulmonary complaint ending in death. Moreover, it is a confirmation of those experiments made upon dogs to elucidate this difficult problem. They were made to inhale an atmosphere containing the sputum of tuberculous patients distributed by a steam atomizer. (See *Virchow's Archiv*, vol. 74, p. 393, not vol. 48, as given in Clapp's book.)

The patient, a young man twenty-three years of age, had lost his mother by consumption. Two years before the onset of his fatal illness, he had suffered for four months from what he considered a cold, which left him without a trace. I first saw him in Sept., 1879, complaining of a cold, which had developed five months previously. The disease was, however, a spotted tubercular trouble of a considerable portion of the upper lobe of the left lung, and to a less extent of the right lung. The symptoms indicated a graver disturbance than the physical signs considered alone would have justified. On a subsequent visit he brought a pet dog with him, which excited my interest by the cough which troubled it from time to time. On inquiry he narrated the following : This was the third dog that he had owned since his sickness. The two predecessors had died after becoming affected with a cough which very closely resembled his own, and that during the paroxysms they at times, as he himself, vomited. All of these animals had been well at the commencement of his proprietorship in them. It was his habit to take the dog to bed with him, and to sleep with it nestling in his arms, its face and snout being turned toward him. In this position it of necessity inhaled his breath, and the atmosphere evolved during his coughing spells, which, at times, were frequent during the night. The first of the dogs was a black-and-tan terrier ; the next a King Charles spaniel ; and the third a Scotch terrier, if my memory serves me. It has always been a source of regret, that he was unwilling to part with his last dog in the interest of scientific observation and a solution of this vexed problem. The young man died, Mar., 1880, but this last animal survived him, though troubled with a cough and having lost flesh and strength.

I have been tempted to repeat the experiment thus performed by this patient, but have not deemed it wise to do so in my hospital service, and in private practice I have not met the right patient. Some of the readers may find the means for the repetition before long. I would suggest that care be taken to select a vigorous animal, and to give it sufficient out-door exercise to maintain its health. If possible, I should advise sacrificing it if sickness should occur, instead of permitting a natural death.

The following clinical cases have some interest also.

Nearly ten years ago I saw a young married woman who was affected with a slow pneumonic infiltration of one lung. The disease began in the upper part of the lower lobe, and gradually spread until the lung became consolidated at the end of seven weeks. During this whole time the only sputum was that ordinarily characterizing pneumonia. Then the process remained nearly stationary for a year. At the expiration of this time she became much fatigued in consequence of a long journey, and in three weeks rapid ulceration and breaking down of the lung terminated in death. Her father was ill with pulmonary phthisis in the same house for some time previous to her sickness. Her husband, a physician, acquired a cough, as he supposed, from exposure to cold in making the arrangements for her funeral. But this was but the inception of a fatal phthisis of the more ordinary variety.

Another group comprises a mother, daughter, and son.

I saw the mother and daughter but once, in consultation with the attending physician, Dr. M. Fleming. The family had no inherited tendency to phthisis. The girl had been affected with hysterical paralysis and other manifestations of this protean disease. A physician who saw her had advised her going to St. Luke's Hospital. While there she was treated in a general ward, in which, according to her account, phthisical patients had their beds. At the time of going she was stout and plump, but shortly began to lose flesh. She remained until December, and then returned home. Soon after she began to have a cough. At the time of my visit, in the middle of May, 1880, she had a large cavity, with thin walls, in the upper lobe of the right lung. Her mother at this time, aged fifty, had spots of tubercular disease in the right upper lobe, some of these being in a state of incipient softening. In her case she had been taken with a cough some time in December. During the autumn she had been considerably prostrated in strength, in consequence of nursing a daughter through typhoid fever, contracted in the Catskill Mountains. Later a son, who at the time of my visit was well, also contracted the disease, and within a few months of one another they succumbed to the malady.

Some of the value of this case is destroyed by a doubt which attaches to the priority of the disease in the mother and daughter. In May the physical signs showed far more advanced trouble in the daughter, but the information about its time of inception of the cough in the two is indefinite. The daughter, however, ran down rapidly before leaving hospital, and before cough was a notable event. The case of the son presents a greater probability of contagion.

Still another recent observation is of interest.

R. S., who died Feb. 23, 1882, of phthisis after a two years' illness, was during the last three months confined to his room. After his death the room was occupied by his brother-in-law and his wife. On May 2, 1882, I saw this gentleman in consultation, he having acute phthisis of the left lung, with a good deal of pneumonic infiltration and rapid ulcerative disturbance. The disease began, according to his account, about five weeks after his brother-in-law's death.

I would add to these the following groups, seen within the last three months.

A young man, engaged to a girl in whose family there was no phthisical taint, became consumptive and died. She was much with him during his illness, and shortly before his death became affected with a cough, which later terminated in death. Her sister, who was much with her during her illness, soon became affected with a cough, which proved chronic, for it had lasted six years at the time of her visit to me during an exacerbation. This was due to a new extension of the disease to the upper part of the left lung, it having been previously confined to the right, as I learned from friends' account of previous medical examinations.

The next group comprises five persons.

The first, a young man sixteen years of age, died of phthisis after several months' illness, four years ago. A sister, aged twenty-two years, who had been much with her brother, after the lapse of a year showed evidences of the disease, and died two years ago. The mother then became ill, but there was an apparent interval between the daughter's death and the commencement of her sickness of some months. Yet, as her daughter says, she had a wheezing in her chest before her sister died, for which she used poultices ; it is not improbable that the disease was present already. She died August 5, 1882. The first that I saw of the family was when called to examine one of the three surviving daughters, eighteen years of age. She had had repeated hemorrhages from the lungs for three weeks, and on examination showed evidences of phthisis at the apex of the lung. Whilst writing this account still another sister has called on me to ask what her sister had better do, and, as the result of a request on my part that I might examine her lungs, I find that she has decided trouble at the right apex, though ignorant of it. These two sisters were much with their mother while ill. There was no hereditary tendency to phthisis on either side of this family.

In addition to these, I have lately examined a young woman who became ill with her phthisis shortly before her mother's death from this disease. Recently, at Bellevue Hospital, a woman ill with consumption had just become affected at the time of her husband's death from this malady. A gentleman a few days since complained of his misfortune in losing the services of a valued clerk, owing to his having fallen a victim to phthisis. On inquiry he said that the man had been closely attentive to his wife, who had recently died after a two years' illness.

The reader can find a number of striking cases collected in Dr. De Musgrave Clay's little work, published in Paris in 1879, or a smaller series, mainly from this source, in the work by Clapp, of Boston.

I add these cases to those which have been published by others, hoping that they may draw attention to this matter, which is one of great importance. I know that many people can be exposed for long periods of time without injury. But the question is, does phthisis spread by the reception of tubercular poison (bacillus?) by those favorably disposed to it, who would otherwise have escaped? In the wards which I visit at Bellevue Hospital are subordinates and attendants who have been there for years, notwithstanding that numerous phthisical patients have lived and died there. This, however, proves nothing more than that those persons have not taken the disease. As an illustration of the want of weight which should be accorded to these negative facts I will use the following illustration:

Some soldiers go through a campaign without injury, whilst others are killed. One might say that bullets, etc., will not kill, because these soldiers escape. Moreover, the disease is slow in its progress, and it is only after a considerable time has elapsed that one can trace the connection; and when several members of a family have died in succession, this is explained on the basis of an hereditary taint. Every physician must have noticed the strong predisposition which some individuals exhibit to diseases known to be contagious. So much so that they at times fall victims, whilst the cause of the poison escapes. I saw this exemplified in the late outbreak of typhus in this city. The physicians under the Board of Health who examined the cases, the physicians who had charge of the cases, the ambulance drivers who removed them, escaped. Cases were for a time in Bellevue, Charity, Roosevelt, Mt. Sinai, St. Vincent's, the German, and other hospitals, yet only three physicians in this city and Brooklyn contracted the disease; one each at Bellevue, the Homœopathic, and at Flatbush hospitals. These physicians were not as much exposed as others.

In one instance a nurse, who had charge of several cases in one family, where the disease had been contracted by two of the children standing near an ambulance, which removed a person sick with typhus, without having the disease herself, conveyed it to a man who occupied a room belonging to the family with which she boarded. She slept during the day on a lounge, on which this man would lie when he came home tired from his work. An attendant in the Small-pox Hospital, contracted the disease, though she had not been in contact with a patient, or the clothing of a patient. She simply came in contact with others who had attended patients sick with typhus, though these did not sicken with the disease. I knew of but one case occurring in the better-to-do class of people, and that was in the person of a merchant whose store is on Broadway. He said that he had seen no one sick with the disease, allowed no peddlers or tramps in his store, always rode in the elevated cars to his home, which was in a part of the city where the disease did not prevail. He contracted the disease by a minimum of exposure at an unknown time, and in an unknown manner. The nurse who took care of him and the physicians who visited him, did not contract the disease, though unprotected. I have chosen typhus fever as an illustration because of its admitted contagiousness, and of its usual requirement of rather prolonged exposure, to show in a clear light the necessity of a third factor beyond the individual and the poison, which, for the lack of a better name, I call predisposition, or, better still, a suitable condition of the system. It may be that with phthisis to a greater extent than with typhus, a prolonged exposure acts only by allowing the poison of the disease to take root, because at some time during this prolonged exposure, it meets with the favorable conditions for its growth. Had I not made careful inquiry, I should have remained ignorant of the majority of the facts here presented. I presume that the hurry incident to practice prevents many physicians from becoming cognizant of similar instances. These cases and others recorded in the literature of the subject have impressed me sufficiently to lead me to give some words of caution upon the continuous use of the same room, and to prohibit in the main the use of the same bed. Also to insist upon those waiting upon phthisical patients having plenty of out-door air and exercise. As regards the phthisis being conveyed to dogs, I believe that amongst the people there must be a superstition, that if the disease can be transmitted to an animal then the person having it may escape. At least I have been asked on several occasions questions which seemed to point in that direction.

THE ETIOLOGY OF CONGENITAL TALIPES EQUINO-VARUS.

By H. W. BERG, M.D.,

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LITTLE as we know concerning the etiology of disease in general, this knowledge will appear relatively extensive, when compared with what is known of the etiology of congenital diseases and deformities. This is probably owing to two reasons: 1st. The difficulty of discovering any cause of disease in a stage of life which ought to be purely physiological and normal; and 2d, because the conditions of fœtal life are especially difficult of investigation. It is not, therefore, surprising that congenital talipes equino-varus, although so common a deformity, has its etiology still unsettled. Nor is this owing to a lack of study of the deformity; for the frequency of its occurrence, together with the seeming simplicity of the subject, has attracted all orthopædic writers, so that there are few who have not either advocated one of the more or less generally accepted theories, or advanced a new one.

To attempt a review of all of these, with the arguments for and against them, would be to perform an almost useless task. For many of these theories are mere theories, having no foundation in well-observed facts. But, on the other hand, we can not properly proceed to the study of the subject, without first becoming familiar with, at least, the opinions held at the present day by leaders in this department of surgery.

Orthopædic writers may be divided into two classes as regards their views concerning the causation of congenital talipes equino-varus.

1. Those who consider the deformity as the result of a mechanical force acting upon the fœtus in utero.

2. Those who consider it the result of disease (intra-uterine), whether of the osseous, muscular, or nervous systems.

Let me first consider the mechanical theory. This was far more popular among older writers than among those of the present day. It still, however, has prominent advocates; among whom may be mentioned Parker, of London,[1] Vogt,[2] Volkmann,[3] Luecke,[4] Banga,[5] and Kocher.[6] By these authorities it is assumed that, for various reasons, the foot, in certain cases, is maintained in one fixed position, by the pressure of the walls of the uterus, for so long a time that it becomes permanently fixed in this position. This theory would thus account for all the forms of club-foot, and this general applicability is its only merit. For, apart from the fact that the fœtus in utero is not an inert mass, but a living being, which moves about in its surrounding fluid, and does not maintain its feet in any fixed position, the theory has this objection, that other parts of the body, which, under similar circumstances, should be subjected to a similar pressure, are not found, with equal frequency, to have acquired this false position. Permanently flexed knee-joints, for instance, should be, according to this view, a very frequent instead of the rarest possible deformity. Indeed this should be a more frequent deformity than club-foot; for this abnormal pressure, which is supposed to take place when the liquor amnii is small in amount, must be equal on all sides of the fœtus, so that those joints to which are attached the longest levers should be most cramped. Were the mechanical theory true, "clubbed" legs and "clubbed" thighs should be more frequent than "clubbed" feet.

This theory is supposed to be strengthened by the fact that fixation of a joint for a lengthened period produces a limitation of the joint's motion. This is certainly so; but in club-foot we have nothing similar to anchylosis. There are no adhesions to overcome. On the contrary, motion as far as is possible in the deformed position of the joint, is good, and as soon as we rectify the deformity by tenotomy, the motion of the joint becomes unrestricted.

The second class of theorists consider the deformity as due to some pathological change affecting the child in utero. To this class belongs the large majority of authorities upon this subject. The secret of its popularity lies in the fact that there are many points of similarity between congenital and non-congenital cases of club-foot; so that it is tempting to ascribe to the former the well-known etiology of the latter. "Really," said Little, as long ago as 1839, "we can clear up the etiology of congenital club-foot by comparing it with that of non-congenital cases. These are caused, first, by paralysis; second, by reflex contracture." And in 1853[7] he again expressed the opinion that congenital club-foot was due to a "disturbance or disease of the nervous system."

Following the lead of this distinguished pioneer in orthopædic surgery, we find most of the succeding writers ascribing congenital club-foot to one or more of the causes which are known to be active in the production of non-congenital cases. The fallacy of such reasoning is evident when we consider the various possible pathological causes in some detail.

First, then, a portion of the older writers held that congenital talipes equino-varus was due to a primary displacement of the bones, resulting, secondarily, in a tonic contracture of the muscles, whose points of origin and points of insertion had been approximated. Hence, an operation on the muscles was not indicated. But as soon as dissection had demonstrated that, while old cases of congenital club-foot, which had used the deformed feet in walking, did indeed show considerable displacement and deformity of the bones, in the new-born infant with club-foot such displacement was not seen, it was understood that in the former cases the displacement of the bones was due to the use to which the deformed limb had been put. Stromeyer recognized this, but went even further and considered the muscular contracture as the primary condition in the pathogenesis of the deformity. In this he was a disciple of Delpech,[8] who taught that a muscle could be the seat of permanent spasm, and thus disturb the relation of bones to each other. We now know that this view is faulty, and by no one was this so well shown as by Volkmann.[9]

He proved, without doubt, that tonic retraction of healthy muscles does not occur, whether the opponents be healthy or paralyzed ; and further, that the structural shortening of the muscles is due, not to contracture, but to growth while the foot remains in its deformed position. However erroneous the opinions of Stromeyer may have been, they still led to highly beneficial results. For it was the conviction that the muscles were the primary cause of the deformity that led him to perform the operation of tenotomy, the benefits of which in the relief of various deformities have been so invaluable to the human race.

Prof. A. Luecke[4] has advanced an original theory, which places him among those who consider the osseous structures of the joint as the seat of primary lesion in club-foot. This writer has observed the fact that at a certain period of life in utero all fœtuses are club-footed. He considers that this early deformity is counteracted by the constant movement of the child in utero, which serves to grind off the articular surfaces of the joint in such a manner as to enable the child's foot to assume the normal position before birth. "If, however," he reasons, "the movements of the fœtus are restricted from any cause, this attrition of joint-surfaces will not take place, and the child will be born deformed." However ingenious this theory may be, it is not very tenable, for granting that at a certain stage of fœtal life the joint-surfaces are opposed to each other at such an angle as to cause the feet to be "clubbed" (which has not been proved by dissection), then surely motion in the deformed position will only serve to increase the faulty direction of the articular surfaces. Indeed, it is motion and use of the deformed feet which make them worse after birth; why should the same mechanical force be followed by a different result during fœtal life? But apart from this we know that in non-congenital cases want of use does not so change the articular surfaces as to cause permanent club-feet. In spastic paralysis, for instance, when the feet have been held in equinus for years, tenotomy is followed, temporarily, at least, by a return of the foot to its form and function. This fact is stated by Dr. Paul Ruprecht, of Dresden,[10] who has had much experience in tenotomy for this cause. I should not have drawn particular attention to this theory, were it not that Prof. Luecke's paper recognizes two important facts:

1st. That the fœtus at a certain period of its existence is club-footed.

2d. That children born with talipes equino-varus have a deformity of the whole extremity, although he has radically mistaken the character of this deformity. The importance of these two facts will be seen later on, when we develop our view of the etiology of club-foot.

In thus discussing the theories which make the bony structures the primary seat of deformity in this form of club-foot, we have unavoidably mentioned those by which the muscles, apart from their connection with the nerves, are considered as the determining factor in the etiology of this affection. No one can suppose that the contraction of the fibrous tissue around the joints—plantar fascia,—ligaments, etc., is any thing but secondary, for being non-contractile (in an active sense) they merely undergo stretching or structural shortening to suit any position of the joint which is long maintained.[11]

It is therefore only left us to review those who account for this deformity by a disturbance or change in the peripheral or central nervous system. Among these are included Barwell,[11] who believes talipes varus to be due to a want of development of the motor tracts; Broadhurst,[12] Little,[7] Malgaigne,[13] and Bonnet (de Lyon),[14] according to whom this deformity is due to some functional or organic disease of the nervous centres; Rudolphi,[15] and Louis Bauer,[16] who rather incline to the belief that a functional disturbance of the nervous system, perhaps reflex in character, can cause a " spasmodic action " of certain muscles, which, they claim, should it continue for even a short time, while growth is going on in the healthy opponents with the rapidity which is characteristic of this stage of life, would soon disturb the even balance between the opposing groups of muscles, thus rendering the deformity permanent. In addition to these authorities, we have Wm. Adams,[17] who discusses all of the principal theories of the etiology of club-foot. He considers that the theory of arrest of development, which we shall take up separately, has been satisfactorily disproved by dissection, nor does he believe in the mechanical theory, but rather vaguely seems to give his adherence to the theory of the nervous origin of club-foot, admitting the possibility of a paralytic affection of the muscles (ant. tib. group) in early fœtal life, giving rise to this deformity. Prof Sayre[18] devotes very little space to the discussion of this question. He thinks, however, that most congenital cases are paralytic in their origin.

A classification of these various opinions upon a pathological basis, would be as follows :

1. That club-foot may be caused by paralysis or spasm of a set of muscles due to cerebral disease.

2. That it may be caused by paralysis or spasm of a set of muscles due to disease of the spinal cord.

3. That it may be due to a reflex disturbance of the nervous centres.

Taking up the first, we find that cerebral lesions may cause deformity in two ways:

a. By the mere paralysis of muscles (deformity resulting from their inactivity—Volkmann[9]).

b. By the secondary contracture which follows paralysis of cerebral origin.

Were the deformity of talipes equino-varus or simple varus due to a cerebral lesion and paralysis resulting therefrom, we should expect to find not a single group of muscles but a whole side paralyzed; which certainly is not the case in children with congenital club-foot, apart from the fact that congenital talipes occurs so often in both legs. As for a local lesion of the cortex, such as a premature ossification of certain parts of the skull, causing pressure and producing this deformity, the fact just stated, together with the healthy condition of most of these children, is opposed to any such theory. We have here none of the symptoms of cerebral disease, such as convulsions, exalted reflexes, etc.

A very commonly assigned cause has been a lack of development of a portion of the motor tracts of the brain. This is accompanied by a secondary lesion of the lateral columns of the cord, and that portion of the anterior columns designated as the columns of Tuerck, and gives rise to a condition known as spastic paralysis (Erb) or tetanoid paralysis (Seguin),[19] in which talipes equinus and sometimes equino-varus is the deformity of the feet. In addition to this is cited the fact that the anacephalus monsters, and children with hydrocephalus, have so often been found to be club-footed.[11] But in all of these cases the deformity is due to contracture of groups of muscles, and we have not only club-foot but a general rigidity and spastic condition of the muscular system, an exaltation of all reflex action, and an evident lack of mental development varying in degree from only a slight amount of slowness in cerebral action, to utter absence of intellect as in anacephalus monsters. But surely these children cannot be likened to the intelligent and otherwise healthy children with congenital club-foot, in whom there is no disturbance of the nervous system ; while objectively, the foot has a different appearance from that in cases of a spastic nature. The principal point of difference being a peculiar movement of the toes in cases of the latter class, somewhat resembling the athetoid movements of the fingers described by Hammond,[20] which is never present in congenital club-foot. Finally, to reason that because talipes occurs with the above conditions, these are necessarily the cause of all cases of congenital talipes, is as illogical as it would be to consider that since abdominal pain occurs with peritonitis, all abdominal pain is due to peritonitis.

Paralysis due to disease of the cord is a fruitful cause of non-congenital club-foot, hence the frequency of the opinion, that this is the most probable cause of congenital cases. In children the affection of the cord most commonly productive of paralysis, is poliomyelitis anterior, and to this disease can be traced a large majority of the non-congenital cases of club-foot of every variety. It is well known that after this paralysis has lasted for a certain time, contractures of opposing and non-paralyzed muscles take place, and the foot assumes a deformed position. As to the mechanism of the production of this deformity I would again refer to the excellent paper of Volkmann[9]; but the fact that it does take place is undisputed. And what can be more tempting than to suppose that congenital club-foot is due to a similar inflammation of the anterior horns of the spinal cord? But a careful examination of the condition of the muscles paralyzed by poliomyelitis anterior, compared with that of the muscles which should be assumed to be the seat of paralysis in congenital club-foot, does not support any such theory.

First, a mere objective examination of a case of congenital talipes and one of infantile paralysis reveals various points of difference. In the first the leg is fat, well nourished, and of normal surface-temperature, showing no signs of the muscular atrophy and cold surface which are characteristic symptoms of the second. Again, in many cases of infantile paralysis other muscles besides those of the leg are the seat of paralysis, while in congenital club-foot this is never the case. Lastly, the electrical examination shows a radical difference. Muscles paralyzed by anterior myelitis do not react to the faradic current, while the galvanic reaction is altered in quantity or quality, or both. I have carefully examined six cases of congenital talipes equino-varus, all under sixteen months of age, and found normal faradic reaction, while there was no change from the ordinary formula to the galvanic current, although it was somewhat difficult to pick out the individual muscles, and the quantity of the reaction was slightly diminished. But certainly in all of the muscles the closure of the negative pole produced a greater contraction than that of the positive. The above cases had not yet begun to walk, and were therefore best fitted for examination.

It might be claimed, however, that the regenerative power of the fœtus is so great as to produce a recovery from the lesion in most cases, so that after birth the neural symptoms above stated would be absent, although the temporary paralysis had been sufficient to fix the foot in a deformed position. But when we consider that contracture does not take place in poliomyelitis anterior until months after the paralysis, and that, should recovery take place early, as often happens even after birth, the contracture would not occur, we will readily understand that the deformity cannot be explained in this way. A more extensive study of this subject will lead to the conviction that no lesion similar to poliomyelitis anterior is the cause of congenital club-foot.

Finally, we come to consider the possibility of this congenital deformity being due to a spastic contracture of the muscles, either from disease of the spinal cord or of reflex origin. It is well known that, theoretically, a primary lesion of the lateral columns of the cord (crossed pyramidal tracts) should produce a spastic condition of the muscles obtaining their nervous supply from that portion of the motor tract in which the lesion is located, and I presume that it is to such a lesion that the present upholders of this theory would refer some cases of congenital club-foot. But it is by no means generally acknowledged that a primary sclerosis or degeneration of the lateral columns of the cord ever occurs in children; and even were such a condition possible, the cases would resemble those of spastic paralysis due to secondary degeneration of the lateral columns, which we have already discussed, and to which, as we have shown, children born with club-foot bear no resemblance.

For the same reason it is necessary to exclude from the etiology of congenital talipes a similar spastic condition, supposed to be the reflex expression of some distant pathological condition of the general system, even were we inclined, in the light of recent discussion, to acknowledge the possibility of such a reflex spastic paralysis.[21, 22]

In concluding this review of the most important of the existing opinions concerning the etiology and pathogenesis of congenital talipes equino-varus, which I considered necessary to a proper understanding of the subject, I would call attention to the fact that none of the foregoing theories very well conform with the acknowledged hereditary element in the etiology of this affection. The mechanical theory, its principal point being an accidental position of the fœtus in utero, certainly does not ; and were we to acknowledge any disease of the nervous system as the cause, the laws of heredity would lead us to expect the appearance of other nervous diseases in the same family, in addition to the one causing the deformity in one or more of its members.

An examination of the family history of the last forty-six cases of congenital talipes equino-varus which have appeared in Dr. N. M. Shaffer's service at the Orthopædic Hospital, shows only one case in which a neurotic element occurred in the history, and this was an "anterior myelitis" affecting another child of the same family. In two cases there was a family history of phthisis. In the remaining forty-three cases the family history was healthy.

All of the theories hitherto discussed, with the exception of the mechanical theory, have considered this congenital deformity as the result of a pathological process. I shall endeavor to present, in a new light, a view which, although feebly advanced by some authorities from time to time, has never been supported by well-observed facts, and has, therefore, gained no adherents. This theory supposes that the deformity of pes equino-varus is due to an *obstruction in the normal development of the feet, during some period of* *intra-uterine life.* This would seem, at first, the most natural direction in which the thoughts of the inquirer concerning this subject would turn. For in this way we can account for many congenital deformities, such as hair-lip, spina bifida, congenital cysts, congenital herniæ, etc. But analogy only hinted at a similar etiology for club-foot without proving it, which was not enough to force conviction, even when combined with the negative proof that all other theories were insufficient.

It has only been in the last few years that the study of human embryology has advanced sufficiently far to enable us to bring positive arguments in favor of this theory of obstructed development; which, as we shall see, does not necessitate, as Adams[17] seems to have thought, any radical change in the individual bones and muscles which form the club-footed extremity.

It is not surprising, then, that after an extensive search in the latest literature of the subject I found no use made of this advanced embryological knowledge to establish the etiology of talipes equino-varus. But lately, in consulting Billroth[23] upon some other subject, I happened to turn upon the chapter on the etiology of club-foot, and found that he strongly expresses his convictions in favor of this latter theory, as explained by recent embryological data, although he does not clearly define either the theory itself or the data upon which it rests..

The fact that in early fœtal life (sixth to seventh week) the sole of the foot is turned in, in the varus position, was acknowledged by many of the earlier writers, such as Isidore Geoffry St. Hillaire,[24] and Wagner,[25] although so little was known concerning human embryology, that even this fact, now so well established, was denied by such authorities as Cruveilhier,[26] Malgaigne,[13] and Little.[7] But those who had observed this fact, considered that an arrest of development at this early stage (third to fourth month, Wagner; sixth to seventh week, Geoffry St. Hillaire) would perpetuate the early fœtal position of the foot.

In exactly what changes the normal process of development consisted, and how the foot gained its normal position, they did not understand. It will here, then, be instructive to study the main facts in the process of the development of the lower extremities, dwelling upon those points only which are of importance in the present discussion.

As early as the third or the beginning of the fourth week of the life of the human embryo, we can distinguish four small budding projections, which correspond to the future extremities of the fœtus (Kölliker,[27] Barwell,[28] Dalton,[29] Gray[30]). These projections or tubercles are due to an increased rapidity of growth over a small, limited area of the external blastodermic layer or epiblast, thus forming an external fold, into which shoots a projection from the outer lamina of the mesoblast or somato-pleura, from which the voluntary muscles of the chest and abdomen also originate. At first these tubercles project horizontally from the body, but as they grow they adapt themselves to the curve of the body of the fœtus.[31] Their relative position at this early period does not exactly correspond to that of the upper and lower extremities later on. For the upper pair of tubercles are relatively low down, about the middle of the fœtus, while the lower pair are relatively high up. But as the embryo progresses, the cephalic and caudal ends grow less than the intermediate portions, and the extremities come to occupy their relatively normal position. The excellent plates of the fœtus at this early stage, in Kölliker's work, already cited, as well as the rather diagrammatic representations of the development of the chick embryo in the works of His and Forster and Balfour, will illustrate my remarks.

For our purpose it will be unnecessary to study the origin of the component tissues of these tubercles or embryo extremities. It will be sufficient to follow the gross, objective changes which these extremities undergo in their development, and this study can be best carried on by a comparison of specimens of the human fœtus at different stages of embryonic life. This I have been, to some extent, enabled to do by means of the specimens of the human fœtus at the New York Hospital Museum, aided by a study of the authorities upon the subject.

At about the fifth week the clefts between the fingers, and later, those between the toes, are formed, and we are enabled to see that the extensor surface of both limbs looks upward and corresponds to the dorsum of the fœtus, while those which will be, later on, the radial and tibial borders are directed toward the cephalic extremity of the embryo, (specimens 877, 1351, and 1527, N. Y. H. M.), and the works of Prof. Humphrey[31] and Albert Kölliker.[27]

At about this period a differentiation takes place along the axis of the rudiments of the extremities, and a mass of the original tissue or "blasteme," as it is called in the translation of Kölliker's[27] work, is converted into cartilage with its perichondrium, thus forming a continuous cartilaginous skeleton for the extremities (Kölliker[27] and Barwell[28]). Soon, however (sixth or seventh week), shallow grooves begin to appear externally, marking the position of the future joints, the distal segments being first distinguished. The first appearance of a joint is seen soon afterward, when whitish lines appear, crossing the cartilage opposite these grooves, and by a process of softening and liquefaction of the original cells, according to some authorities, among whom is Barwell, the joint cavities are formed. Here again the most distant joints (those of the phalanges) are formed first, the others following in the reverse order of their proximity.

Kölliker is uncertain whether the joints are formed by a process of softening (*ramolissement*), and we cannot here enter upon the views of different authorities upon this point, but he agrees with the opinion that the skeleton of the extremities is originally one continuous cartilage, and that as the joints are formed, it becomes divided into the different segments of the extremity. The first joint appearing from the sixth to the eighth week, and the last at the end of the third or beginning of the fourth month.

If, then, we examine a fœtus at the beginning of the third month, we find that the elbow and knee joints have already been formed. But the extensor surfaces of the upper and lower extremity no longer look in the same direction; for, whereas before both patella and olecranon were directed backward, and later on outward, now the knee is directed outward and principally *forward*, while the elbow is looking outward and principally *backward*. That is to say, the extremities have altered their primitive position in some manner, and assumed the one which they maintain through life. The rotation at this early period is not yet complete, but soon becomes so in the ordinary process of development.

This movement is not characteristic of the human fœtus, but has been observed in many of the vertebrates. Forster and Balfour,[32] in their researches on the embryo of the chick, found that although at first the angles of both the joints, corresponding to the knee and elbow, look outward and somewhat backward, yet on the eighth day the elbow looks directly backward and the knee forward. Prof. Humphrey,[31] who has carefully studied this rotation of the extremities, describes it in the following manner: "In the primitive position the extensor surface of the two limbs is directed upward or dorsally, the radial and tibial borders being directed forward. In this position the two extremities are homologous in their parts. Subsequently, by a quarter turn, to some extent participated in by the pelvis, the extensor surface of the hind limb is directed forward, and the tibial border now looks inward. By a quarter turn in the opposite direction, to some extent participated in by the shoulder, the extensor surface of the upper limb is directed backward, the radial border therefore looking outward. At the same time pronation occurs in the forearm and hand, whereby the palm is brought in contact with the ground." This view is the one generally adopted, although Dr. P. Albrecht, of Kiel,[33] holds a somewhat different opinion, into which we cannot now enter, and in answer to which Prof. Humphrey wrote the article from which I have quoted.

The cause of this rotation which, as I shall soon show, is of the greatest importance in the study of the etiology of congenital club-foot, is unknown. Kölliker remarks that it cannot depend upon muscular power, for the muscles have not yet appeared when this rotation has begun. He thinks, however, that the phenomena of growth which take place in the members themselves as well as in the adjacent regions, play an important part in this rotation.

The movement does not take place suddenly, but beginning early, at about the second month, is to a great extent accomplished by the fourth month; but not completed until the fifth and beginning of the sixth month.

A studyof the specimens in the New York Hospital Museum, as well as those in the Wood's Museum of Bellevue Hospital, will show the progressive changes in the position of the extremities at different periods of fœtal life. These specimens were examined in the glass jars in which they were mounted.

As will be seen from the table, not only is the leg as a whole rotated outward during early fœtal life, but this outward rotation is accompanied by an exaggerated varus, and

NUMBER OF SPECIMEN.	MUSEUM.	AGE.	APPEARANCE OF EXTREMITIES.
877	N. Y. H. M.	4 weeks	No joints apparent in limb buds. Extensor surface of both limbs (upper and lower) directed dorsally. Limbs project perpendicularly from body of fœtus.
1351	N. Y. H. M.	6 weeks	Peripheral joints and those at knees, apparent. Limbs applied to sides of fœtus. Extensor surface of both limbs directed outward.
884	N. Y. H. M.	2 months	All joints present; lower extremity rotated *outward.* Thighs flexed upon abdomen; legs at about right angles with thighs, crossing each other and applied closely to body of fœtus. Knees look outward. Feet in *equino-varus* position. Soles on a concave line with inner border of leg. Arm rotated inward. Radius directed toward the head.
1472	N. Y. H. M.	3 months	Leg still rotated outward. Knee looking outward and very slightly forward. Feet in exaggerated varus position. Abdomen very protuberant.
1201	Wood's M.	4 months	Legs flexed upon thighs at less than right angles. Inward rotation of thighs about half complete. Feet in equino-varus position; inner border raised, and deeply concave so as to conform with outline of abdominal wall. Elbows directed outward.
1203	Wood's M.	5 months	Inward rotation of legs very far advanced. Knees look forward and slightly outward. Varus of feet slight. Equinus no longer present. In upper extremities elbows look more backward, but here, as in lower extremities, right side appears further advanced in rotation than left.
1204	Wood's M.	6 months	Rotation complete. Knees look forward. Feet almost as straight as in new-born children. Inner border of feet raised more than normal, and with arch of feet exaggerated. No trace of equinus. Rotation of upper extremities complete. Elbows look backward and very slightly outward.
890	N. Y. H. M.	7 months	Rotation of legs complete. Tibiæ directed inward. Soles of feet look downward. Inner borders not abnormally raised.

later an equino-varus, position of the foot, which diminishes as the rotation of the lower extremity to its normal position progresses, although even when the rotation of the leg has been entirely completed, some of the varus deformity of the foot still remains; and a careful observer will notice a very slight tendency to varus even in the newly born child, which gradually disappears as the foot develops after birth. This fact is confirmed by Prof. Dalton. In addition to the varus deformity we often see in fœtuses of the second, third, and fourth months an equinus. This is not always present, and soon disappears in the course of the normal growth of the foot. We find, then, that at an early stage of fœtal life, talipes varus or equino-varus is physiological. Ordinarily, however, in a healthy fœtus little of the outward rotation of the leg or of the varus of the foot remains after the end of the sixth month.

Further study and observation will lead to the conviction that this physiological varus is dependent upon the primary outwardly rotated position of the leg in early fœtal life. To this conclusion we are led by even slight study of the relative positions of the thigh, leg, and foot during intra-uterine life.

As soon as the joints are formed, we find the thigh rotated outward as far as possible, and flexed upon the body. The leg is flexed upon the thigh, but not completely, for this is prevented by the extreme outward rotation of the thigh, which brings the inner border of the leg in apposition with the abdomen of the child. We have, then, the inner border of the thigh and the tibial border of the leg pressed against the abdomen of the fœtus, the legs crossing each other a little below their middle. (This position is typically shown in specimen 884 N. Y. H. M., described in the table). All of the intra-uterine pressure, therefore, is thus brought to bear directly upon the outer border of the thigh and leg, corresponding to the fibular border of the leg, and also upon the dorsum of the foot. The result of this is that the foot is rotated in and extended (equino-varus) until the sole is almost on a line with the inner border of the leg, and lies against the body of the fœtus, while the dorsal surface of the foot is on a convex curved line with the outer border of the leg, to adapt itself to the concave wall of the uterus. This, I believe, is a stage in the normal development of every healthy fœtus ; and were the extremities to remain in this position all children would be born club-footed. But nature provides against this by the inward rotation of the extremity, which gradually takes place, carrying the leg away from its position against the abdomen of the fœtus ; and when this rotation is completed, we find the extensor surface of the thigh flexed and in relation with the body of the child, while the legs are flexed upon the thighs, the inner or tibial borders facing each other. Now the soles of the feet lie against the uterine walls and the intra-uterine pressure is exerted directly upon them. This produces extreme flexion of the foot upon the leg, together with an outward rotation of the foot ; this movement, from the constitution of the ankle-joint, accompanying extreme flexion. Thus is antagonized the varus or equino-varus which has existed hitherto. It is evident, then, that upon the completeness of the internal rotation or torsion which takes place in the lower extremity, depends the rectification of the early varus of the foot. Should this rotation not take place at all, or be incomplete, the foot will continue to maintain its early relation to the body of the fœtus and uterine walls, and the child will be born more or less club-footed. If this be so, we should expect to find in club-footed children that the extremities are rotated outward. And this we do find upon examination. In all of the cases of congenital club-foot (equino-varus), which I have seen since my attention has been directed to this subject, I have found, that the thigh and leg as a whole were rotated out, and the tibia bent inward at its lower part so that the feet were approximated to each other in addition to being in the clubbed position. All this is seen to be the result of the non-rotation of the leg. This can be confirmed by any one, upon a young child suffering from this deformity. At the Orthopædic Dispensary my colleagues have confirmed these observations.

Here I wish to call attention to the fact that no extraordinary amount of intra-uterine pressure is necessary to produce this deformity, its cause being not pressure, but as we have seen a non-rotation inward or a retarded rotation inward of the lower extremities. This I maintain in spite of the accidental occurrence of pressure-marks on the feet of a few club-footed children, and notwithstanding the observation, in an equally small number of cases, that the birth of a child with pes equino-varus was accompanied by a deficiency in the amount of liquor amnii. Both of these facts have been advanced as proofs that club-foot was due primarily to an abnormal amount of intra-uterine pressure by such observers as Volkmann,[3] Luecke,[4] Vogt,[2] Banga,[5] and Kocher.[6] Vogt himself acknowledges, however, that such an "*ad oculos*" demonstration of abnormal intra-uterine pressure is rare. On the other hand, almost every case of congenital equino-varus will show evidence of the condition to which we have ascribed the deformity: namely, non-rotation inward of the lower extremity. This is best measured by the direction in which the knee looks. In the large majority, and perhaps all, of the children with congenital equino-varus, the knee will be found looking outward, and only slightly forward.

Luecke[4] states the direct opposite of this, and says that in these children the thigh is rotated inward, and the knees form a genu valgus. That this statement is in contradiction to the actual position of the legs in congenitally club-footed children who have not yet walked, any one may convince himself by personal observation, and I can account for it in no way but that it was an unintentional error on the part of this writer.

In support of the theory, then, which I have here developed, we have:

1st. The fact that the leg is rotated outward in all cases born with this deformity.

2d. That during embryonic life a similar deformity is present, as long as the leg has not yet completed its inward rotation.

3d. That this view is perfectly compatible with the otherwise healthy condition of most of these children.

4th. That this theory readily harmonizes with the hereditary element in the history of talipes equino-varus congenitus. For it is easily conceivable that the same lack of developmental energy which characterizes one member of a family may be present in other individuals of the same family.

5th. That upon this basis can easily be explained some complicated cases of club-foot, which would otherwise appear as inexplicable anomalies: as, for instance, the occurrence of club-hand and club-foot in the same patient. Here the hand has undergone a retardation of development as well as the foot.

Adams quotes an extreme instance of this, which he does not attempt to explain. The case is that of a child with equino-varus of both feet; knee and hip joints stiff; radius turned inward and facing toward the head (this, as we have seen above, is the early fœtal position). The elbow and shoulder joints were in a condition similar to that of the knee and hip (page 221, *op. cit.*). Here, evidently, the outward rotation of the upper and the inward rotation of the lower extremities have not taken place. Both have been kept in the primitive position—a position which does not admit of much motion. Torsion not taking place, even to a slight degree, the joints have never been exercised, hence their stiff and semi-anchylosed condition. Upon no other basis could such a concurrence of deformity in the upper and lower extremity have been so well explained.

In this connection I would describe a very interesting specimen in the Wood's Museum collection, numbered in the catalogue as 1220. "A fœtus showing general deformity." "Hands and feet clubbed." This is an eight-months' fœtus, in which the rotation of the lower as well as the upper extremities has not taken place. The result being that the extremities have retained the early fœtal position with regard to the body of the child; the legs are flexed at about right angles with the thighs, while the inner borders of the thighs, legs, and feet are pressed closely into apposition with the abdomen, thus causing an exaggerated equino-varus deformity of the feet, and stiffness of the knee, and especially of the hip joints, owing to the causes above stated.

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TWO CASES OF DIPHTHERITIC SPINAL ATAXIA.

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THE subject of paralysis consecutive to acute diseases of various nature has received ample notice at the hands of many writers on nervous affections. Especially have the paralytic derangements which follow diphtheria been accurately and thoroughly described ; it would therefore be entirely superfluous to reiterate what has been told so many times, and we shall consequently not enter into a description of diphtheritic paralysis in general, but refer more particularly to a certain peculiar form of these accidents.

The fact that the differential diagnosis between a diphtheritic paralysis of the lower extremities simulating locomotor ataxia, and a genuine case of tabes, may become difficult, seems to have attracted the attention of only a few writers upon this subject. In nearly all books the articles on locomotor ataxia, or those on diphtheritic paralysis, entirely ignore the possibility of the two diseases being mistaken for each other, and it is precisely on account of having met with this difficulty ourselves that we have been led to call attention to the cases which will be found further on.

As early as 1836 Orillard,[1] in Poitiers, mentioned ataxic symptoms as late sequelæ of diphtheria, and Trousseau, Sée, and Maingault had remarked the unsteady gait in certain cases of diphtheritic paralysis, and had even attributed it to muscular anæsthesia. Eiseman[2] and Jaccoud,[3] however, were the first persons who seemed to separate the motor paralyses of the lower extremities following diphtheria from ordinary paralyses, and to classify them as cases of diphtheritic locomotor ataxia, and it is with them that the name diphtheritic ataxia originated. Eiseman and Jaccoud, however, do not agree, inasmuch as Eiseman considers all cases of anomaly of movement following diphtheria as ataxia, whereas Jaccoud makes a distinct differentiation between paralysis and ataxia. Afterward English writers, Grainger Stewart,[4] Foot,[5] and others, described cases of this disorder, and in late years several more have been added to the list, so that at present the existence of such a disease is undisputed. Eulenburg, who, in the first edition of his textbook, speaks of an "apparent incoördination," in his later editions distinctly acknowledges the existence of a real diphtheritic ataxia. Brenner[6] speaks of diphtheria as being able to cause incoördination of movements without atrophy of the muscles, and with normal electrical contractility of both muscles and nerves. His is probably the best and most lucid classification of the nervous sequelæ of diphtheria.[7] He divides the anomalies of movement following this disease into three groups. In the first group he classes those cases in which the interference of motion is due to pure ataxia. In the second, those in which it is due to an ataxic paralysis, characterized by a paresis of certain muscular groups, and the more complete paralysis of certain others. In the third, finally, those which are due to a true paralysis. The cases which come under the first heading, namely, those where the anomalies of motion are due to pure ataxia, are quite rare, and therefore those recorded further on will be of interest.

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[1] Maingault, *Arch. générales*, 1859, Oct., p. 388.

[2] "Die Bewegungs Ataxie," p. 192.

[3] "Les paraplégies et l' ataxie du mouvement," p. 631.

[4] *Edinburgh Med. Jour.*, 1870, p. 988.

[5] *Dublin Journal of Med. Science*, 1872, p. 176.

[6] "Untersuchungen auf dem Gebiet der Electrotherapie", ii, p. 207.

[7] *Petersburger med. Zeitschrift*, Bd. x, Heft 4 and 5, 1866.

The facts about ataxia in general are so well known that it is not necessary to review them here; it is only requisite to state that we can divide all disassociation of movements into spinal and cerebral (includes cerebellar). We desire particularly to call attention to this point, for the cases of cerebral ataxia following diphtheria are not at all rare; in fact, in many cases of diphtheritic paralysis there exists a certain amount of ataxia, associated with obtuseness of intellect, dizziness, headache, and cerebral symptoms in general. These cases, then, have, by various authors, according to the preponderance of the paralysis or of the ataxia, been described as cases of diphtheritic paralysis or diphtheritic ataxia. Langner,[1] under the supervision of Prof. Berger, reports several cases of cerebral ataxia following diphtheria, and the majority of cases published as diphtheritic ataxia come under this category.

Case i.—J. H., twenty years of age; grocer. Temperate habits; had always been well until the 17th of December, 1879, when he caught cold by exposing himself to the night air, and the next day complained of pain in his throat and difficulty in swallowing. These symptoms augmented in severity until the 22d, when he was obliged to take to his bed. He remained in bed until the 25th, during which time he had medical attendance. His physician told him he had "ulcerated sore throat," and that it was full of "blisters." He claims not to have had any fever. Remained in the house until the 2d of January, and then feeling quite well, and not having any more pain in his throat, he went to work again. Two weeks after he noticed that his voice had become nasal in character, that frequently while swallowing he spluttered the food out through his mouth and nose. He describes it as though a cramp of the muscles took place, which threw the food, especially fluids, back. At times particles of food also entered the larynx. These symptoms, however, only lasted a few days, and then disappeared. Thereupon he complained of weakness of the lower extremities, and following this, of the hands. The condition of patient when he came under our observation, March 1st, was as follows :

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[1] Inaug. Dissert., Breslau, 1878.

Patient is tall, strong-looking, and well built. Tongue, mouth, throat, and uvula normal. Swallows without any difficulty, has good appetite, and digests well. Circulatory, respiratory, and genito-urinary systems healthy.

Voice slightly nasal, but he claims that it was always so. In examining the upper extremities we found the sensibility to heat and pain normal. He does not localize perfectly, and there is a retardation in the appreciation of sensation of several seconds ; thus, when his eyes are closed, he does not feel the prick of a pin on the back of his hand until fully two seconds after he has been so pricked. The muscular power does not seem to be diminished, and the movements of his hands are not noticeably affected ; but the more delicate movements of the fingers, movements requiring great precision, are executed with unsteadiness. He cannot button his clothes, or pick up any small object. If be endeavors to pick up a needle from the table, he only succeeds in doing so after many awkward attempts. The lower extremities also retain full muscular power, but he walks very unsteadily and has a marked ataxic gait. His knees knock together at intervals while walking. The sensibility to touch, heat, and pain is normal, except in the soles of the feet. When he walks he says he cannot feel the floor distinctly, but that it feels as though he were walking on a sponge. The unsteadiness in walking is increased by closing his eyes. If he stands with his legs spread apart he is more steady, but when he brings them together, he must sustain himself to avoid falling.

Muscular development good.

Patellar tendon reflex entirely missing. Achilles reflex not obtainable. Reaction of both galvanic and faradic currents normal. All cerebral symptoms, as dizziness, headache, vomiting, anomalies of speech and intelligence, were wanting during the entire course of the disease. Patient was treated by sulphate of strychnine pills, $\frac{1}{60}$ grain each ; one pill four times daily. Under this treatment he gradually recovered, until May 1, 1880, when, with the exception of the patellar reflex, which was still absent, he was entirely restored to his normal condition. In May, 1882, I again saw patient, and after this lapse of two years found the *patellar reflex present*, but weak. Otherwise patient is and has been perfectly well.

CASE 2.—H. R. came to consult us the beginning of June, 1881, and we then made the following notes in our case-book:

Family history is, with the exception of one living brother, who is idiotic, uninteresting.

Previous history: Patient was, with the exception of a short cough and occasional headache, both of which he has had since childhood, well until a short time ago. He served through the entire Franco-Prussian war, and was subjected to great hardships and deprivations. He, however, did not seem to have been affected by these, and remained in good health until last March, when he caught cold, had a sore throat, and remained in bed eight days. He says that his throat was covered with "white blisters." About fourteen days after getting out of bed, he noticed that his voice had become nasal in character; also, that he had difficulty in swallowing, especially so when he attempted to swallow liquids, as these were frequently thrown back through the nose, or, getting into the larynx, gave rise to violent spells of coughing. This condition lasted about three weeks, when he noticed quick-shooting pains in both arms and legs. These pains were very severe, and prevented him from obtaining any sleep. A short time thereafter, he began to feel unsteady on his legs, and the floor did not appear solid to him. He says it felt as though it were sinking from under him. He could hardly walk at all in the dark, and walking up stairs was very difficult at all times. Then his hands became affected, first formications, and then numbness of the tips of his fingers supervening. He did not feel objects that he held in his hand, but was cognizant of their presence only by watching them. Frequently let cups and saucers fall from his hands; this occurred particularly when he turned his eyes to something else. Had great difficulty in buttoning his clothes. These symptoms have increased in severity up to the present time.

St. Pr.—P. is a tall, well-built individual, of healthy appearance. Complains of sharp shooting pains in the extremities, but these he says are not as severe as they formerly were. Cannot stand with closed eyes, as he becomes dizzy and would fall if he were not held. Has a feeling as though he were standing on a very thick carpet. Cannot walk in the dark. Muscular force of legs normal. His gait is decidedly jerky; lifting his foot high from the ground, he throws it violently forward, and then brings it sharply to the ground.

Patellar tendon reflex entirely absent.

The muscular force of the upper extremities is also undiminished. The muscles are not atrophied. Has a feeling of numbness in his fingers. With his eyes closed, he cannot distinguish between a pin and a penholder. He can distinctly feel the prick of a pin, but cannot localize it well. Cannot button his clothes without great trouble. Pupils react well. Eyes normal. Reaction to both electrical currents normal. The treatment employed was: sulphate of strychnine, three times daily, $\frac{1}{60}$ of a grain, internally; and, three times a week, a descending galvanic current of sixteen Léclanché elements, along the spinal cord.

August 1st. Patient is somewhat improved. He still cannot walk in the dark. There is a decided improvement in the hands, as he can now button his clothes and dress himself unaided. He claims that they still, however, feel somewhat numb. Patellar tendon reflex still absent.

I then lost sight of patient, and did not again see him until January, 1882, when I had opportunity to re-examine him, and found him fully recovered; able to walk without any trouble. No apparent ataxia, either of hands or feet. *Patellar tendon reflex normal.* Says he never felt better, and that he can walk long distances, and is again able to work at his trade, which, being that of watchmaker, requires fine and precise work.

In these cases, therefore, there being only an incomplete history of diphtheria, and the patients coming under observation after all diphtheritic symptoms had passed away, and at a period when none but tabetic symptoms were present, the possibility of mistaking the disease may be appreciated; the second case particularly, as it occurred in a man who had committed excesses of various kinds, and been exposed to manifold deprivations. There can be no doubt whatever as to the existence of the ataxia, the unsteady gait, the planting down of the heels, the throwing, jerky movements in walking, the unsteady and irregular movements of the hands, are probably sufficient as proofs of its existence. The only question yet remaining which admits of any doubt, is whether the ataxia in these cases can be termed identical with that existing in tabes dorsalis. Certainly, considering the absence of all symptoms which are generally present in cerebral ataxia, we are not warranted in searching anywhere outside of the spinal cord for an explanation of this incoördination.

Dizziness, headache, vomiting, anomalies of speech, or cloudiness of the intellect were not present during the entire course of the disease. On the other hand, the symptoms which were present, such as the formications, the dizziness when standing with closed eyes, the gradual course, first the lower and then the upper extremities becoming affected, and, above all, the anomalies of reflex action, the absence of patellar reflex, render it almost a certainty that the anatomical site of the lesion does not differ from that found in progressive locomotor ataxia, although, considering the course of the disease, and the rapid convalescence, we certainly cannot think of a sclerosis, but must attribute it to some more ephemeral lesion, if we have the right to apply the word lesion at all.

A very important feature of these cases, and one to which very little attention has been paid in cases of diphtheritic ataxia, is the absence of patellar tendon reflex, with its subsequent return. Rumpff,[1] in reporting a case of pure spinal diphtheritic ataxia, of which I will mention the salient points, speaks particularly of the absence of patellar reflex. So also does Langner.[2] Rumpff's patient was a girl, nine years of age, who had a severe attack of diphtheria, and who, during the course of the disease, had difficulty in speaking and swallowing. Four weeks subsequently, paresis of accommodation, insufficiency of the recti interni, and paralysis of the soft palate. After fourteen days she became easily fatigued, had a staggering, unsteady gait, and the more minute movements of the fingers were hampered and executed with difficulty. Reflex of patellar tendons absent. Weakness of extremities. After another lapse of fourteen days, the heels are brought down suddenly, the feet thrown forward, and the skin reflexes almost absent. Muscular force normal. Ataxia of lower as well as upper extremities. Gradual improvement, and after four months power of coördination was again present. The tendon reflexes, however, remain absent for five more weeks.

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[1] *Deutsches Arch. für klin. Med.*, 1877, vol. xx.

[2] Inaug Dissert., Breslau, 1878.

Autopsies have not been frequent in diphtheritic paralysis on account of the rarity of a lethal issue during this condition, and formerly the result of the few *post-mortems* which were made was always a negative one. This, however, need not surprise us when we consider that the aid of the microscope was rarely called into requisition, and that when it was, it was done without the use of the staining materials which are now at our command, and which are of so much service to us in histological and pathological research.

The supposition of Bretonneau, that the paralysis is only a local extension of the diphtheria, is to be cited solely for its historical interest.

Trousseau's theory, which was also supported by Maingault and Sée, that the paralysis is due to systemic poisoning, the blood becoming vitiated by the absorption of the diphtheritic virus, becomes improper to sustain normal nutrition and the normal excitation of the nervous centres, *ergo* the paralysis, certainly at first makes a favorable impression upon the mind ; it has now, however, become entirely untenable. This theory of poisoning has been strongly contested by Prof. Gubler, who observes that sometimes cases of ordinary sore throat, without any specific character whatever, are followed by paralysis of the soft palate. The simple œdema which occurs in most all of these cases would, however, be sufficient to account for this local paralysis. Whether the paralysis be due to vitiation of the blood or not, the question which will naturally propound itself to us, is whether there is any nervous lesion, and if so, where and what that lesion is.

This can be best answered by briefly reviewing the facts now known. The present state of our knowledge, according to Dr. Magne, is as follows:

In 1862 Charcot and Vulpian in a woman deceased during a diphtheritic paralysis of the soft palate, found evident changes in the motor nerves supplying this part. In 1869, Lorrain and Lépine mentioned a similar case, and Liouville has observed an analogous change in the phrenic in a case of diphtheritic paralysis of the diaphragm. Bühl, in 1867, noticed a marked alteration in the roots of the spinal nerves, the cord itself being unaffected. He found the spinal roots and ganglia thickened, discolored, and softened; the neurilemma and the interstitial substance diphtheritically infiltrated. His theory is that the paralysis is caused by this exudation, and if it is absorbed the case ends in recovery, but if, on the contrary, it is changed into connective tissue, the normal state is not restored. Bühl also found capillary hemorrhages in the brain.

R. Maier, in 1870, found microscopical changes which corroborate those seen by Bühl.

Oertel has noticed capillary hemorrhages in the rachidean as well as in the cerebral dura mater; also in the sheaths of the nerve-roots and of the peripheral nerves. In the anterior cornua of the cord, he observed an increase of nuclei and small hemorrhagic infarctions. He also found larger hemorrhages in the brain, cord, and peripheral nerves, and found the central canal occluded by a cellular exudation.

H. Weber[1] supports the view that the paralyses originate by means of a change which is unknown to us, and which is as yet undiscoverable, starting at the periphery of the nervous system (the seat of the diphtheria) and being propagated to the spinal cord in an analogous manner, as in traumatic tetanus, the propagation takes place from the seat of injury.

Pierret[2] in examining the hardened spinal cord of a man deceased during a diphtheritic paralysis, which in the fresh condition was of normal appearance, found a number of changes. The dura mater was adherent, partly thickened and roughened. Upon section the diphtheritic changes became apparent. He found a meningitis, and an exudation in the meninges similar in all points to the pseudo-membranes in the larynx. Vulpian, however, who examined three other cases subsequently, failed to corroborate this.

A more recent investigator of the subject is Déjérine,[3] who in five cases found the same kind of lesion. He found in all his cases a parenchymatous degeneration of the anterior roots. He found constant light changes in the gray substance of the anterior horns. He considers the whole as a slight parenchymatous and interstitial poliomyelitis anterior, and looks upon this as the primary change in diphtheritic paralysis. The alterations in the nerves and in the muscles are, according to his view, only secondary trophic changes. The various sites of the changes, then, as they have been found in the divers cases, would also explain the diversity of the paralytic symptoms. Thus, to explain the paralysis of the soft palate, the tongue, and lips, we must place the seat of lesion in the medulla oblongata. So again in cases where there were anomalies of sight and hearing accompanying the paralysis, the origin of the optic and auditory nerves were affected. In a case where Pierret found the changes in the lumbar region, the subject had been paraplegic. We have not been able to find any reported autopsy of a case of pure diphtheritic ataxia, therefore are unable to say whether the seat of lesion is in the posterior columns of the cord, as from our cases with their tabetic symptomatology and their long ensuing absence of patellar reflex, we would be led to suspect.

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[1] *Virchow's Archiv*, xxv.

[2] *Gazette des hôpitaux*, No. 2, 1877.

[3] Déjérine : Recherches sur les lésions du système nerveux dans la par. diph. *Arch. de physiol.*, 1878, p. 107.

As regards the prognosis and treatment there does not remain very much to be said. Most cases end in recovery, and are not of very protracted duration. The treatment employed in all reported cases has been the use of the sulphate of strychnine, internally or hypodermically, the use of the galvanic current along the spine; or both of these measures combined. Whether the disease is curtailed in its course by these measures, or whether it is self-limited, would be hazardous to say, as the cases from which we are forced to draw our conclusions are not sufficiently numerous.

NOTE ON CRANIO-CEREBRAL TOPOGRAPHY. ILLUSTRATED.*

By E. C. SEGUIN, M.D.

THE surgical anatomy of the head, with reference to its contents, has been developed with remarkable completeness within the last ten years, chiefly by the researches of Broca, Bischoff, Heftler, Turner, and Féré, by which cranio-cerebral topography has been firmly established as a branch of practical anatomy. Acting upon the data thus obtained, Broca, Lucas-Championnière, Weir, and others have succesfully trephined for the relief of aphasic and paralytic symptoms, The location of many convolutions and fissures of the cerebrum can be accurately mapped out upon the surface of the skull, or even upon a living head, by the projection of certain lines and measurements from certain points thus obtained, as well as from some natural landmarks.

For the projection of these lines, the head is placed in a particular position, as can be easily done when we operate upon a bare skull; but which can also be approximated when we deal with a living subject, either sitting up or lying in bed. The skull or the shaven head should be so placed and held that a line passing from the alveolar process of the superior maxilla just at the insertion of the teeth, and through the lowest part of the occipital bone, shall be horizontal. The greatest care should be used to determine this line—the alveolo-condyloid line or plane of Broca,—for it is upon it that all other projections and measurements are based. In the annexed figure the skull is represented as resting upon the alveolo-condyloid plane, 1–1.

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* Reprinted from Gross' "System of Surgery."

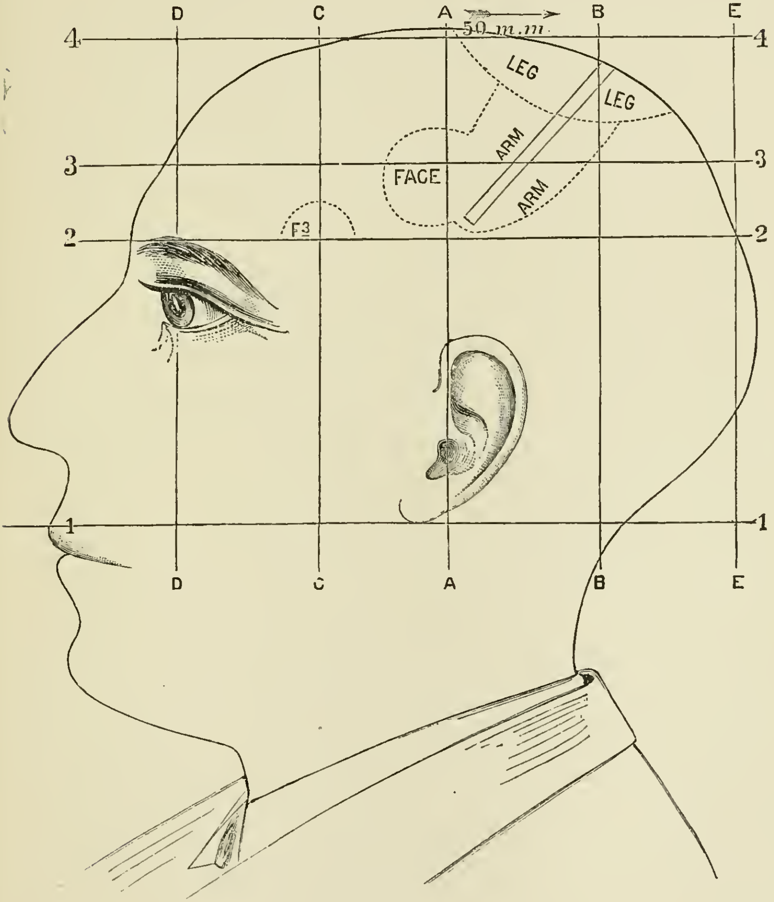

FIG. 1.
Topographical lines applied to the external contour of the head.

Next, from the alveolo-condyloid line a vertical line, or one exactly perpendicular to the first, is drawn through the external auditory meatus. At the top of the head this line —the auriculo-bregmatic line,—A-A, indicates the bregma or true vertex, which is an important landmark, one which should be traced with ink or carmine upon the shaven scalp. Upon the top of the head or skull an imaginary horizontal line, 4–4, parallel with the alveolo-condyloid plane, is projected, and upon this line, 4–4, we measure backward 50 mm. and draw a second vertical line, B–B, parallel to the auriculo-bregmatic line. At the place where this line strikes the convexity of the head is the Rolandic point, R, under which, in average heads, lie the upper or posterior extremity of the fissure of Rolando, the upper ends of the ascending frontal and ascending parietal convolutions, and, within the longitudinal fissure, the paracentral lobule; in other words, the upper part of the motor area of the cerebrum, that which quite probably controls the volitional movements of the legs. The Rolandic point, thus determined, should, on a living subject for operative purposes, be properly marked upon the shaven scalp.

A third horizontal line is next to be drawn from the external angular process of the frontal bone, parallel with the alveolo-condyloid plane. This line, 2–2, which may be called the fronto-lambdoidal, as its posterior extremity will usually pass at or near the upper angle of the lambdoidal suture, serves the purpose of determining the situation of some important parts. In the first place the line 2–2 passes about 5 mm. above the upper border of the squamous suture, and under this line, mostly parallel to it, are the anterior two thirds of the fissure of Sylvius. Secondly, at about 5 mm. above and behind the intersection of lines A–A and 2–2 is the inferior extremity of the fissure of Rolando bounded by the ascending frontal and ascending aprietal convolutions. In the third place, upon this line, 2–2, at a distance of 18 or 20 mm. behind the external angular process of the frontal bone, is the folded part of the posterior extremity of the third frontal convolution, or Broca's speech-centre, marked F³ on the diagram.

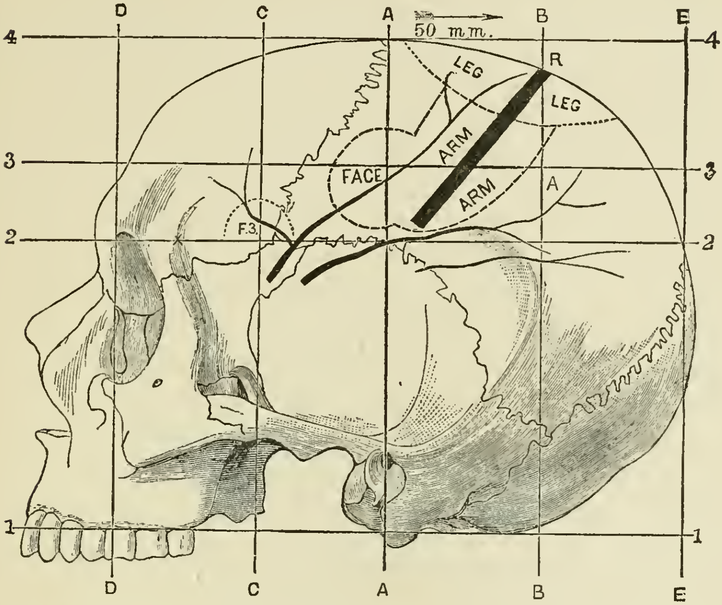

FIG. 2.

Topographical lines applied to Henle's skull. Location of the Rolandic fissure, motor centres, and branches of the middle meningeal artery shown.

Having exactly determined and marked the situation of the Rolandic point at the top of the skull, and the inferior termination of the fissure of Rolando above the ear, these two points are connected by a line which is represented darkly drawn upon the diagram. This, the Rolandic line, is the guide for nearly all operations intended for the relief of traumatic spasm or paralysis, since under it and near it lie the so-called motor centres for different parts of the body on the opposite side, as determined by experiments upon monkeys and dogs, and by numerous post-mortem examinations made in cases of tumors and other limited lesions of the brain.

As indicated by the dotted lines on the diagram, the motor zone or centre for the lower extremity of the opposite side lies about the Rolandic point, making an allowance of at least 10 mm. to either side of the median line for the interval between the two hemispheres. It also includes the paracentral lobule within the longitudinal fissure; and Dr Seguin is led to believe, from observation in cases of cerebral tumor, that this part is preëminently the centre for the leg. Below this, reaching quite down to the fronto-lambdoidal line 2–2, is the motor area of the upper extremity. Forward of this, between the auriculo-bregmatic line A–A and the line C–C, is a part of the second frontal convolution, which probably has connections with the facial muscles of the opposite side. Finally, at F^3 is the speech-centre of Broca, which, although not now regarded as the only speech-organ, yet plays an important part in the simpler mechanism which produces language-motions.

Other relations of interest are the apex of the sphenoidal or temporal lobe a little beneath the line 2–2, and at about 10 or 12 mm. posterior to the external angular process of the frontal bone; the situation of the occipito-parietal fissure almost immediately under the posterior end of the line 2–2, at its junction with the vertical line E–E, which also indicates the posterior extremity of the cerebrum; the anterior end of the brain is marked off by the vertical line D–D. Furthermore, for certain purposes, the limits of the central ganglia of the brain may be estimated as follows: their superior limit is indicated by a horizontal line or plane drawn at a level 45 mm. below the vertex, line 3–3 of the diagram; their anterior limit, which corresponds to the head of the nucleus caudatus, is traced by the vertical line C–C; and their posterior limit, or hinder end of the thalamus opticus, by the vertical line B–B. Lastly, it may be stated that the angular gyrus—a part of the cortex which recent autopsies would seem to connect with vision—lies not far from the point of intersection of the lines B–B and 3–3, at the point marked A on the diagram. This, in the living subject, is a little below and behind the parietal eminence.

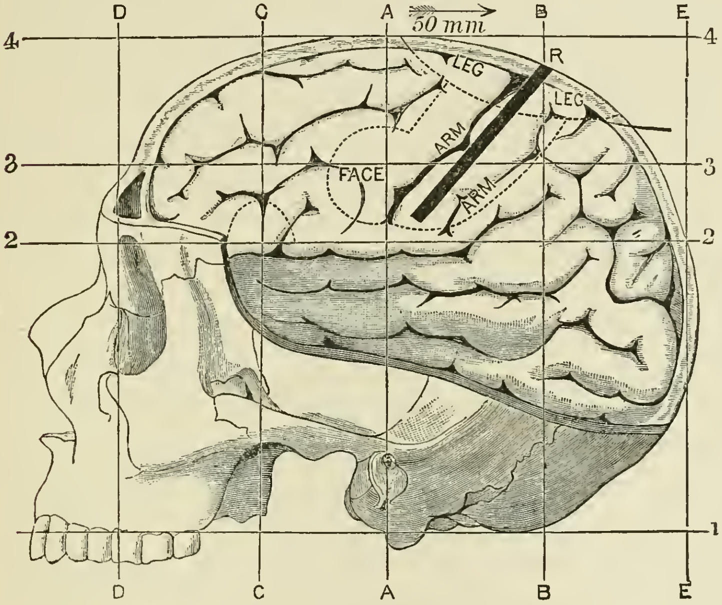

FIG. 3.

Topographical lines and landmarks projected on the convolutions, the apparent non-conformity of the Rolandic line and fissure being due to perspective. Henle's skull.

The location of the middle meningeal artery, which so often furnishes the blood which compresses the brain after various injuries of the head, is surgically considered of great importance. The course of the two principal branches of the artery is approximately indicated upon the diagram by the branching lines drawn on the anterior inferior angle of the parietal bone. In the living subject, the main trunk of the artery would be found under the horizontal line 2–2, at a point a little posterior to the speech-centre, about 30 mm.

behind the external angular process of the frontal bone, and in front of the beginning of the fissure of Sylvius. It passes obliquely upward and backward almost immediately over the whole of the ascending frontal convolution, from 5 to 10 mm. in front of the Rolandic line. The inferior branch of the artery is nearly horizontal, and almost exactly overlies the fissure of Sylvius.

Upon the shaven head of a patient, seated in a chair or lying in bed, the principal landmarks and relations above defined can be mapped out with a great approximation to accuracy by the use of two rulers, or even by one, to mark the alveolo-condyloid plane, and a card-board cut so as to stand astride the skull in the auriculo-bregmatic vertical. A light wooden apparatus could be easily made to indicate these two lines, while the remaining measurements could be taken with a tape, and the points marked with carmine or black ink.

The practical utility of these anatomical data depends upon an acceptance of modern physiological teaching upon the subject of the functions of the brain. The experimental and pathological evidence now accumulated in favor of the connections of the " centres " marked on the diagram and certain peripheral parts, and between the whole of the motor area, and the whole opposite side of the patient is, according to Dr. Seguin, convincing, and leads him frequently to a very accurate topographical diagnosis in medical cases.

The following operations may be referred to as illustrative of the utility of the laws of cerebral localization and of cranio-cerebral pathology :

Broca, in 1871, in a case in which aphasia and paralysis followed a severe lacerated scalp wound, trephined over the left third frontal convolution, or speech-centre, found pus, and slightly relieved his patient.

Lucas-Championnière, in 1874, trephined a man in whom coma, partial right hemiplegia, convulsions, and, as shown during convalescence, aphasia resulted immediately from a slight cranial injury. There was only a slight scalp scar to guide him, but he came in contact with splinters and blood from a fracture existing below the point of apparent injury, and saved his patient.

Hueter, of Greifswald, in 1879, in a somewhat similar case, trephined the skull, ligated the middle meningeal artery, and cured his patient. In another case in 1870 he was equally successful.

Courvoisier trephined, in 1878, a child two and a half years old, who, after an insignificant wound in the left temporal region, had right hemiplegia, coma, and palsy of the left side of the face. He found a fissured fracture, and pus outside the dura mater, as well as a large quantity under it. The operation was followed by recovery with weakness of the right side.

Dr. R. F. Weir, of New York, in 1882, operated at Bellevue Hospital, in a case in which coma and slight hemiplegia existed, the patient being a man who had received a blow on the head. There was no very evident external injury, but guided by the various data of cerebral localization, and proceeding according to the rules of cranio-cerebral topography, the trephine was applied, and a small clot found between the brain and dura mater. On incising the latter the brain was seen to be extensively disorganized, and the seat of copious hemorrhage, which was checked by torsion. Although the symptoms were relieved by the operation, death occurred within a few days.

Up to the present time, so far as I know, there are only three cases in which the rules of cranio-cerebral topography have been applied, from measurements actually made prior to operation. These cases occurred in the practice, respectively, of Broca, Lucas-Championnière, and Weir. There are probably other examples, but I am unable to particularize them. However this may be, there is a large number of recorded instances of relatively or absolutely successful operations performed after cranial injuries, for immediate as well as for secondary effects, without measurements.

The indications for trephining after cranial injuries for the relief of symptoms of cerebral irritation, compression, or disorganization, may be provisionally stated as follows:

1. When aphasia supervenes immediately or within a few days or weeks after an injury of the anterior portion of the head on the left side. It is extremely probable, in the first case, that a clot or bony spicule will be found compressing the speech-centre; and in the second, that an abscess has formed either in the same part or close to it.

2. When simple hemiplegia, or hemiplegia with hemispasm, follows an injury, however slight, in the temporo-parietal region. If the spasm or paralysis be limited to one limb or to the face, the indication to operate is even stronger. Even if the injury be not directly over the motor area, the surgeon is justified in such a case in exploring that area.

3. In conditions of coma after cranial injuries, sometimes without external wound, in which meningeal hemorrhage is the cause of death, the discovery of slight hemiplegia should call for trephining planned according to the rules above laid down, as in Weir's case. Dr. Seguin suggests that a latent hemiplegic state might be discovered, at least in some cases, by an increase of peripheral temperature, as of the fingers or toes, on one side, and by the presence of congestion or of an erythematous blush on one buttock.

4. In the very rare cases in which the paralytic phenomena are found on the same side of the body as the cranial injury, it might be proper to trephine on the opposite side

of the skull in search of hemorrhage or fracture, the result of contre-coup.

5. In chronic epilepsy after traumatism of the head, the indication for operation is present, but it is not a specific indication, connected with the subject under consideration. A lesion of any part of the skull may be a cause of epileptic attacks, irrespective of the motor centres.

Contra-indications to trephining may be thus enumerated:

1. Whenever in apparently favorable cases there are signs indicative of lesion of the base of the brain, such as palsy of several cranial nerves, neuro-retinitis, or Cheyne-Stokes respiration.

2. When hemiplegia is accompanied by great anæsthesia, rendering it probable that the lesion is beyond the motor area, deeper, and farther back.

It should be understood that these indications and contra-indications are formulated from the stand-point of the neurologist. The surgeon, upon general grounds, will doubtless often modify them, and add others.

It is obvious that our knowledge of cranio-cerebral topography is not perfect, and, in a practical point of view, very unsatisfactory. As yet, we have broken only a little ground; the great work remains to be accomplished. In a field so vast as this our progress must necessarily be tardy, and many hands will be required to solve its mysteries.

EDITORIAL DEPARTMENT.

HOSPITAL MANAGEMENT.

It gives us great pleasure to bring to the notice of the general medical public the fact, that in two of our best hospitals steps have been taken toward lengthening the terms of service of the attending staff.

Attention has been repeatedly called in the editorial columns of this journal to the existing evils in hospital management in America, and the almost universal prevalence of short terms of service has been justly deprecated.

Public hospitals serve, or should serve, two ends, both of which are for the public good. The first is, as humanitarian institutions, the welfare of their helpless inmates; and the second is, the advancement of the science of medicine.

To show how a lengthened term of service will conduce to these two ends, which are really one, a brief review of facts is necessary.

In most of our city hospitals a division is made into a medical and a surgical side or service. In some a subdivision of these services is made. Each one of these services, consisting of from fifty to one hundred patients, is under the superintendence of several attending physicians or surgeons, who in rotation assume charge of the entire service. As they are generally four or more in number, and as each generally wants to be on duty once every year, it reduces their term of service to three months or less.

In the first place, the attending has more patients than he can do justice to. When he comes on duty he finds a great many cases convalescent through his predecessor's skill. These cases possess little to interest him. Another lot of cases are those upon which three months' treatment will make but little impression, and these, too, very naturally fail to interest him. The third set of cases are those which enter the hospital during his service, are afflicted with acute diseases, which, whether under his treatment they tend to speedy cure or dissolution, never fail to excite his unflagging interest, and to receive his almost undivided attention. What is true of one attending's actions is true of all. The convalescents, left by one, get well as best they may. The acute cases receive his whole attention, while the chronic cases are passed on from one to another, and from one hospital to another, receiving nowhere the attention they need. What is true of the attendings is true also of the internes. They have too much to do. The mere fact of their having more than they can do well prevents their doing as much as they otherwise might. Following the example of their elders, they devote most of their time to the care of the acute cases, to the exclusion of the study and care of those chronic cases which in later years will form the bulk of their practice.

The existence of these facts, which no man who has had a hospital experience will deny, proves conclusively the welfare of the patient is not subserved by short terms of service and inadequate house-staffs. Neither is the hospital as an educational institution bettered by this state of affairs. The learners in a hospital are first the attendings, next the internes, and lastly the general medical public. The advantage to the attending of a six months' over a three months' service need hardly be mentioned. The service being presumably subdivided, he can spend more time in the study and care of individual cases. He can watch the more protracted cases from beginning to end. He can teach his internes methods of investigating, recording, diagnosing, and treating cases, now impossible. He can avail himself and make available to others countless clinical facts by bedside and lecture-room talk. He can present to the general medical public through journal articles and monographs the result of his experience or original investigations. The staff of internes will necessarily be doubled. This will give so many more young men a hospital experience, which is so essential to future success. They cannot plead overwork as an excuse for dereliction of duty ; better discipline will be maintained, and they will have time for outside reading and study.

It thus seems that a careful consideration of the subject could lead only to the conclusion that a lengthened term of service would result only in good.

The step taken by the attending physicians and surgeons of St. Luke's Hospital, where a six months' service has already been inaugurated, and a doubling of the house-staff asked for, and by the surgeons of the New York Hospital, where a similar change has been asked of the governors, deserves the heartiest commendation of all. Whether the suggestions of the respective medical boards will be carried out by the governing boards, remains to be seen.

NEW BOOKS AND INSTRUMENTS.

The International Encyclopædia of Surgery. A systematic treatise on the theory and practice of surgery by authors of various nations. Edited by John Ashhurst, Jr., M.D., Professor of Clinical Surgery in the University of Pennsylvania. In six volumes. Vol. 1. New York: Wm. Wood & Co., 1881.

Anybody familiar with the eminent success achieved by Billroth and Pitha's collection of monographs on surgical subjects, later on of that of Ziemssen's, and finally of Billroth and Lücke's encyclopædias, will find it very natural that the embodiment of a similar idea in the English tongue was a mere question of time. Careful study of certain parts of this eminently American work—its international title to the contrary notwithstanding—will well repay the trouble. At the head of the first volume appears the well-rendered translation of a treatise by Professor S. Stricker, of Vienna, on "*Disturbances of Nutrition; the Pathology of Inflammation.*" Though some enthusiastic adherents of Professor Stricker's peculiar views on many subjects of normal and morbid pathology have popularized his name to a certain extent on this side of the ocean, yet it will appear odd to many that an undoubtedly indefatigable and original investigator, but one whose views are still opposed to a remarkable extent by the majority of equally earnest workers among his countrymen, should have been selected as the exponent—so to say—of German histological science. The pleasure of reading the lucid exposition of the thoroughly original views of Prof. Stricker will certainly be appreciated by every practitioner familiar with the other side of the many controverted questions, and far may it be from us to forget that many of the novel aspects of old questions as set forth are full of suggestions and possibly fruitful promises. But it is difficult to evade the thought, that the practising surgeon will rather look in a similar work for a concise and impartial exposition of the state of histological knowledge of the day, than for an undeniably able and very interesting but thoroughly *ex-parte* statement of strongly contested and hitherto uncorroborated theories.

On the other hand the selection doubtlessly has points that will commend it. The lack of original investigators among the large number of intelligent men in the American profession has been repeatedly commented on publicly, in journals and otherwise ; and to illustrate the attractiveness of the intimate and subtle machinery of the mental work of an original student, the choice could hardly have fallen on a more interesting personage than Prof. Stricker. Moreover, the tendency of modern science has clearly demonstrated that the object of scientific teaching is not so much the imparting of the simple knowledge of facts, but rather the training of the student's mind and senses in the methods of ascertaining by his own effort, by search, observation, and criticism, the facts included in any special branch of knowledge. And Stricker's treatise certainly possesses many of the features of this very modern tendency. It reads almost like a pleasant story, and its lucid style, the new light thrown on many still dark subjects, bits of historical reference and controversy, add much to the attractiveness of the otherwise dry subject. It would be out of place to enter here on a detailed criticism of the several views held by the author, and it will suffice to mention some leading features of the treatise, together with a few glimpses on such views that are novel and peculiar.

The treatment of the difficult subject of the vaso-motor nerves is excellent, and will be read with pleasure by everybody. The difference between the two forms of active hyperæmia, that *of paralysis* and that *of irritation*, together with the rationale of their vaso-motor influences, namely paralysis of the constrictors on one side and irritation of the vaso-dilators on the other, is well elucidated. The confirmatory vivisectional experiments are extremely interesting. The views held by the author about *internal flowing* and *undulating motion* of the cell-body ; the relations of cell and basement- or intercellular substance, and their interchangeability under certain circumstances, which is sometimes pressed to absolute identity of both, are very attractively exposed. Of the four cardinal symptoms of inflammation, two, viz.: heat and redness are contracted into one conception, that of active hyperæmia. Pain and swelling, not being necessarily and universally met with in inflammation, are discredited as trustworthy symptoms of the process ; and *tissue metamorphosis* is insisted on as being of cardinal importance. The so-called migration theory of suppuration, first brought forward by Cohnheim, is declared as not proven, and in its place the author's own theory of tissue metamorphosis is proposed, with the arguments pertaining thereto. *In nuce* the theory is as follows : Under certain irritative influences the basis substance is converted into cell substance ; the cells return to the embryonic state ; then they divide into ambœoid, movable masses, which become detached from each other and thus acquire the character of pus cells. This theory holds good for all kinds of tissue, and the attempt of applying it to tissues of such divergent nature as the cornea, cartilage, bone, nerve substance, muscle, and connective tissue is carried through with much ingenuity and consistency. The fixed nature of the cells of the cornea, bone, cartilage, and other tissues is seriously questioned ; their essential identity and interchangeableness is vigorously insisted upon, and only one metamorphosis of the living substance is admitted as resulting in a permanent form of tissue, which, however, is devoid of functional capacity, and that is the so-called *fibrillar degeneration* as exemplified in tabes dorsalis.

The theory possessing the undeniable advantage of simplicity, is easily made use of to explain the process of healing by first intention and by suppuration, likewise that of ulceration, and the regeneration of muscular and nerve tissue.

The prevalent systems of classification of pseudoplasms are brushed aside as *mere child's play*, and unworthy of serious consideration, but no substitute is suggested therefor.

As previously mentioned, the translation by Dr. A. Meyer is very good and remarkably free from Germanisms.

Prof. Van Buren's essay on "*Inflammation*" implies not only the necessity of looking at the more practical or clinical aspects of this process, but can, in a manner, also be accepted as an apology for the one-sidedness of the first paper of the series. Otherwise it would appear to be superfluous to go again over ground presumably covered by Stricker's article. In the theoretical, introductory parts of the paper due attention is paid to the views of other pathologists than the Viennese professor, and the desire for impartiality and thoroughness as regards the enumeration of all possible causes and *modus operandi* of inflammation is very apparent. The term "enumeration" is purposely chosen, so far as the arrangement of the matter is concerned.

The leading theories of modern investigators, such as Pasteur, Koch, Ranvier, and others, are noted up to the date of publication. The juxtaposition of the latest information regarding septicæmia with notions belonging to an obsolete period of pathology, is striking. Cold and sudden chilling are mentioned as direct causes of inflammation, though the actual proof falls short of conviction. The illustrative case given with reference to this point is an instance of *post hoc ergo propter hoc*.

Among the mass of useful knowledge, statements occur which must challenge doubt. So it is said that the puerperal state, as such, predisposes to suppuration, and that collections of pus in joints *post partum* lack a satisfactory explanation. The etymology of the Greek affix, "*itis*," used to denote inflammation, is far from being as simple as given by Van Buren. That the uniform high temperature of injured organs, as in subcutaneous fractures or tenotomy wounds, plays any essential part in determining a kind process of healing, is, to say the least, very problematic ; and that even a moderate addition to the habitual use of alcoholic beverages predisposes to suppurative inflammation, is simply not proven. That the practice of inoculating the cornea and conjunctiva with fresh pus to cure pannus is employed *on a large scale* in Germany, will strike most German oculists as a startling statement.

The favorable results after operations for disease, contrasting so markedly with the higher proportion of deaths after primary amputations, are explained by a sort of adaptation to, or tolerance of, operations, in consequence "of a previous training by illness and pain." In contradiction to this, Oberst[1] proved, from the extensive experienee of the surgical clinic of Halle, that many deaths after primary amputation are due, on one hand, to the shock and loss of blood sustained with severe injuries, necessitating immediate operation ; on the other hand, to the fact, that in primary amputations the larger size of the cut surface of generally robust limbs, the quantity of severed vessels, etc., render the successful practice of antiseptic surgery much more difficult than in the smaller transverse sections of the usually emaciated members of patients undergoing secondary amputation. In short, the danger from amputation can be said to increase in a direct ratio with the size of the cut surface.

The author is much more happy in the treatment of the purely practical portion of his essay, and here it becomes evident that the careful and observant surgeon is treading on ground well explored by himself. It is gratifying to read the sound and judicious remarks of Prof. Van Buren about the use or abuse of poultices, so commonly met with ; on local and general blood-letting ; on the practice of incisions and drainage, and nursing in general.

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[1] Die Amputationen unter dem Einfluss der antiseptischen Behandlung, Halle, 1882.

Prof. Alfred Stille's paper on "*Erysipelas*" is a fair model of a terse, condensed, yet withal attractive representation of a subject full of difficulties. Throughout it bears the marks of a thorough knowledge and assimilation of the literature of the matter in hand, and in drawing conclusions the author displays the clear unbiassed judgment of a philosophical observer. Arrangement of matter and style exhibit the skill of an experienced and successful teacher. Signs of careful preparation are met with everywhere. Conclusions are drawn deliberately, sometimes boldly ; and a refreshing critical spirit, untrammelled by the traditions of the shop and worthy of a philosopher, pervades the sentences. The remarks on the general treatment of the malady are replete with good-sense, and form a fit rebuke of the popular chase after specifics, the use of which is only too often the screen to hide mental indolence or incapacity, or both. Among the generally sound and unapproachable statements, exception has to be taken to some. The idea that the ancient treatment of erysipelas by applications of cold water is dangerous, lacks objective support, and is smacking of prejudice. The assertion that *alcoholic stimulants increase the fever* is flatly contradicted by recent observers. The preoccupation in favor of the tincture of chloride of iron, and the detractory opinion held about quinine, stand in strange contrast to the general tone of the essay. The author's abovementioned objection to alcohol, when made by others as to iron, is thus disposed of: "The question is not whether iron raises the temperature, but whether this preparation of it tends to cure erysipelas." That equal success is claimed for quinine, whether administered in small or very large doses, is mentioned as a fact tending to discredit the assertions of its adherents. But Prof. Stillé forgets that this circumstance holds good as to the tincture of iron also. We do not find any mention of the author's own experience with quinine in erysipelas, and therefore conclude not to take his remarks, "*au sérieux*."

In the perusal of this paper time will be well employed.

Dr. Francis Delafield's well-known name is a guaranty of good work, and the wisdom of the author in confining himself to a brief, but pregnant and clear summary of the debated questions pertaining to "*Pyæmia and Allied Conditions*," is very commendable. The embodiment of this conception will best satisfy the demands of the active practitioner for whom this encyclopædia has been admittedly gotten up. The indubitable facts bearing on pyæmia are well and clearly stated ; the doubtful points are characterized as such ; and the decision is left to a jury, composed of all minds interested in unravelling the mysteries of this most terrible enemy of the practical surgeon.

That condensation and terseness may be carried too far, Dr. Wm. S. Forbes' paper on "*Hydrophobia and Rabies ; Glanders and Malignant Pustule*" fairly illustrates. A book of reference ought to present more of the pathology of a well-known morbid process like malignant pustule, than a mere sketch of a sketch. Davaine's discoveries are so well authenticated, and having formed the historical starting-point for myco-pathological researches now occupying the attention of the entire scientific world, would seem to deserve more than six lines of trivial statement.

Henry Trentham Butlin, the author of the disquisition on "*Scrofula and Tubercle*," seems to enter into his subject under the shadow of an unnecessarily gloomy view respecting the state of our knowledge of its pathology. For after providing a palpable substratum to his deductions in the shape of a few well-chosen histories of cases, he manages to extract from the chaos of contemporaneous information a tolerably correct picture of the anatomical and clinical features of both conditions. And since the proper combination of clinical observation with the pathology of disease will in all probability forever remain the only safe and popular way of studying maladies, the author can be said to have tolerably well fulfilled the requirements of his task. Recent investigation is noticed up to the date of publication, and the new aspects of tubercular pathology are set forth in a manner to suggest more exhaustive study.

"*Rachitis*" found a very able exponent in Dr. J. Lewis Smith, of New York, whose long-extended experience in this special field adds much weight to his opinions on practical questions of treatment of this malady. General and experimental pathology are adequately considered, and although the manner is not brilliant, the matter is well selected and perspicuously arranged.

Whatever Surgeon-General Wales may have to say on "*Scurvy*" will be accepted by the profession as coming, "ex cathedra." The editor of the Encyclopædia must be congratulated on the happy choice of this author for this particular theme. An erudite and yet attractive historical introduction at once captivates the interest of the reader ; and the careful arrangement of the quality and quantity of matter, together with the elegance of literary style, co-operate in forming a monograph, complete and well rounded off, and certain to bring credit to both the author and the entire publication.

Professor Verneuil's favorite theme, "*The Reciprocal Effects of Constitutional Conditions and Injuries*," will disappoint those expecting to find a well-finished article. Undoubtedly it has lost much by translation ; and the elegant diction gone, its intrinsic defects are all the more prominent. The reader will save time and avoid vexation by skipping the thoroughly dogmatic, and oftentimes obscure introduction, which would do honor in its pedantry to an ossified German professor of a former period. Much credit is due, nevertheless, to the distinguished French surgeon for having first studied in a systematic manner the relation of injuries to constitutional disorders. Exception will have to be taken to some assertions. It is interesting but altogether doubtful that "the neoplastic diathesis itself is strictly dependent upon arthritism—which is equivalent to saying that neoplastic and cancerous subjects are merely arthritic patients suffering from a special manifestation of the constitutional disease."

The opinion that acute alcoholism forms a positive contra-indication to the employment of anæsthetics, will not be shared by many American surgeons, to whom it is well known that weakly and cachectic patients will stand better a prolonged anæsthesia and much loss of blood, when prepared by a liberal administration of stimulants than otherwise. The parallel drawn between acute alcoholism and diabetes mellitus, in calling the former poisoning by alcohol, the latter *poisoning by sugar*, will at once suggest a further extension of this unphysiological comparison to the state of albuminuria, which might be aptly called *poisoning by albumen.* A strange confusion of cause and effect !

In giving his adhesion to the antiseptic mode of wound-management, the author admits the necessity of modifying his views, as held in the ante-Listerian period ; but he omits to draw a corollary by stating that wounds and injuries, if received at an incipient period of a constitutional disturbance, that is, at a time when the general condition is not yet gravely impaired, will heal as kindly and readily as those inflicted on a healthy organism. And English and German surgeons have demonstrated that under antiseptic cautelæ even the wounds and injuries of the cachectic victims of syphilis, tuberculosis, and scrofula will heal correctly, often by first intention, as long as accidental wound-complications are excluded by well-known procedures. Of course, this advantage cannot diminish the necessity of a careful medicinal and dietetic treatment of whatever constitutional disorder may be present.

Prof. Verneuil's remarks about the curious effect of injuries in calling forth a renewed attack of a long-dormant constitutional disorder, deserve more attention than the subject has been accorded to thus far by the profession. Much to be regretted is the omission of even a single concrete illustration of some rarer forms of this latter curious process. Throughout the essay no reference is made to the author's large personal experience, or to other observers' publications bearing on this subject.

Professor D. Hayes Agnew's paper on the "*General Principles of Surgical Diagnosis*" is full of valuable hints to the young practitioner. Mature judgment and prolonged experience, together with an agreeable style, enable the writer to accomplish his task in an excellent manner.

Dr. Mansell-Moullin's scholarly paper on "*Shock*" contains all that is worth knowing about this curious form of reflex paralysis. The reader is at once put *in medias res*, and etiology and symptomatology form a pleasant introduction to the pathology of the condition, which may be considered to form the focal point of the short, but thorough and well rounded-off essay. The author's great familiarity with the practical side, as well as with the literature of his theme, deserves all the more credit, as every important fact is carefully verified by reference, and thus may serve as an excellent starting-point for a more extended study of the original authors. It would be hard to add to, but harder still to take away from, the paper one sentence without crippling it.

Dr. Wm. Hunt's article on "*Traumatic Delirium and Delirium Tremens*" reads almost like a nightmare. If the editor intended to introduce a bit of medico-comic writing into the collection of serious essays by way of pleasant change, he undoubtedly succeeded very well. A serious criticism of the article seems hardly possible, and a Pickwickian consideration of the florid, almost poetic style of the author would be certainly very amusing, if not profitable. It must suffice to quote a few examples of the peculiar phraseology and metaphorical style of the paper. A "rare form of traumatic delirium" is thus illustrated by the author in a supposititious case: "The surgeon enters some ward in the morning after a terrible accident has occurred, and finds that a victim *of this kind*[1] has just been brought in and laid upon a bed. He is at once recognized by the patient as one in authority. 'How are you, doctor?' he says, in a high voice; 'what have they brought me here for? I'm not hurt! No, sir! Look at that,' and out goes an arm with the force of a prize-fighter delivering a crusher. 'Look here,' and he tries to lift a leg, which his sensorium falsely tells him he has done, although his expression may indicate a vague and passing doubt. 'Why, there is my wife! Molly, what are you doing here? don't cry; what are you crying for? I'm not hurt; go home to the children, and tell them I'll be there to supper, and at the mills to-morrow. Won't I, doctor? Go home!' Soon this great tension gives way, collapse comes on, and by night the patient is in another home than that in which he promised to be. I have never known such a case to recover."

We find after an enumeration of the well-known causes of traumatic delirium, several pathological conditions gone into extensively, *because they do not cause delirium*, as, for instance, snake-bites, bites of "venomous spiders *and* insects," hydrophobia, tetanus, and chorea. A chart exhibiting the temperatures of a case of surgical fever with delirium finds its commentary in a pathetic, but very fanciful quotation from "Rab and his Friends." Burns and scalds also receive a quasi-poetical treatment. "I have seen after deep and extensive burns, what seemed to be a living soul looking out of a crisp, charred, and dead body. I have seen a child thus burnt, playing with toys an hour before its death,'[1] etc., and then follow six lines of appropriate verse.

Delirium tremens is also rhapsodized over in the drastic manner of Dr. Hunt, and is fitly branded as "a fiend, sitting as a cormorant hard by the surgeon's efforts, and devouring all his measures for good." Further on we read that "the peculiar tremors of delirium tremens are very characteristic; if I may so express it, they are loose and free, without spasmodic jerkings; somewhat *jelly-like*, and giving a general idea of instability. They seem to say: 'Prop me up, prop me up; if you don't prop me up I shall die.'" From this the conclusion is drawn naturally and easily, that "propping up" must be the treatment.

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[1] Reviewer's italics.

The article appears like a huge joke, and we must refer the curious reader to the original.

Professor Henry M. Lyman, of Chicago, contributes a very creditable paper on "*Anæsthetics and Anæsthesia.*" The description of the phenomena of anæsthesia is a little masterpiece in its vividity and truthfulness. About two pages of print comprise everything of importance regarding the mortality consequent on artificial anæsthesia. The known proportions of deaths are percentually stated, and the reader is left to draw his own conclusions.

We miss detailed directions for the use of ether and chloroform. So, for instance, the common occurrence of the patient's tongue slipping back on the epiglottis, thus impeding or arresting respiration, and the simple method of projecting the tongue by pushing the lower jaw forward, is not mentioned at all. The disagreeable by-effect of ether in causing very profuse salivation, inspiration of saliva, and occasionally pneumonia, is ignored. The statement of the fact that ether is, chloroform is not, a respirable vapor; that, accordingly, ether can be administered without an admixture of air, but chloroform ought not, is too important and too suggestive to be omitted. We may add, however, that many of these details bearing on the practice of anæsthesia will be found more fully treated in Dr. John H. Brinton's essay on "*Operative Surgery in General.*" This subject being very well known, little opportunity is given to the author to show originality; but every remark shows the sober good judgment of the experienced surgeon. The commendation by the author of the judicious use of stimulants in the shape of alcohol and opium before grave operations, and his candor in advising the use of chloroform as an anæsthetic in cases of lung trouble in preference to ether, is to be noted. The caution not to apply Esmarch's constrictor to a limb when its muscles are shortened, as in flexion, is valuable, for if the distal portion of the limb be afterward extended, serious injury and laceration of the upper structures may ensue. With some surgeons Lister's antiseptic wound treatment is still *on trial*, and Dr. Brinton belongs to these doubters. It is only fair to say, however, that he freely admits the beneficial effect of Prof. Lister's labors in transforming ancient into modern surgery. The repetition of subjects disposed of in former papers could have been advantageously avoided.

A number of well-known manuals would have shown to Dr. Charles T. Hunter how to deliver himself creditably of such an easy theme as "*Minor Surgery*" is. Among surgical dressings the omission of the most popular dressing of the period—Lister's—will create surprise. The manifold modifications of Lister's original conception—each of them useful and applicable in certain cases,—such as Bardeleben's moist jute dressing, Thiersch's salicylic acid and cotton dressing, the iodoform dressing as recommended by Mosetig and Mikulicz, finally Neuber's valuable additions to the chapter on permanent antiseptic dressings, are all ignored; but "Revulsion and Counter-Irritation" affords the author an opportunity of enlarging on such important topics as "Issues"—not forgetting even the "*issue pea*"—and "Setons." Commendable is the mention of *Massage*, and its forms of application, a subject undeservedly omitted from almost all handbooks. We fear it must remain a riddle what the sphygmograph has to do with minor surgery.

It is difficult to find a *raison d' être* for Prof. Christopher Johnston's short disquisition, entitled "*Plastic Surgery*"; for the general remarks based on Verneuil's classification would be a suitable beginning to a treatise on special plastic surgery, and are too ponderous to form an introduction to the slim and threadbare subject of skin-grafting. Dr. Frank H. Hamilton's claim to priority in suggesting transplantation is gone into somewhat extensively, and forms the most noticeable feature of the article.

Prof. John Ashhurst, Jr., concludes the volume with a very able and, *sit venia verbo*, carefully written monograph on "*Amputations.*" A brief historical sketch is followed by a complete enumeration of the causes calling for amputation, after which the apparatus for blood-saving receive due attention. Among the aortic compressors Lister's is preferred by the author. No mention is made of the new method of compressing per rectum the common iliac, or even the aorta, by the aid of a lever. To diminish troublesome oozing after the removal of Esmarch's band, the author warmly recommends the simultaneous application of a tourniquet with the constrictor. The moment the latter is removed, the tourniquet is brought to bear on the main artery, and very little hemorrhage ensues. It is very regrettable that Prof. Ashhurst has seen no evidence yet to make him think animal ligatures in any way better than the hempen or silken ones. The overwhelming majority of surgeons of the present day seem to have been more fortunate in this respect. Notwithstanding the lessons taught by the experience of the last fifteen years, we find the distinguished author still clinging to the traditions of the long-spent pre-Listerian period. Lister and his disciples are set down as followers of "a fashion," and the merits of the antiseptic treatment of wounds are superciliously disposed of in a few sneering remarks, ill becoming an earnest searcher after truth. The testimony of the author's own cases shows that he never gave this method a single trial, and that therefore his belittling of the accumulated experience of the best men of the period—many of them older veterans of the amputating knife than Prof. Ashhurst himself—is narrow-minded and unjust. Whether "bacteria and micrococci are met and destroyed in their efforts to reach the wound, as in Prof. Lister's dressing;" or "by Mr. Guérin's device are mechanically arrested by a huge thickness of cotton wadding, and being unable to get either in or out, miserably perish in its meshes," is immaterial as to practical results. The upholders of the new departure can well afford to smile at such arguments, when they compare the author's 28 deaths out of 100 amputations (4 at least of pyæmia), with the results achieved by Prof. Volkmann in 261 amputations. Among 73 ablations for injuries, 7 (10.5 %) died of collapse or anæmia within a few hours after operation ; of 188 amputations for disease, 4 died of acute anæmia or collapse almost immediately after operation, 1 of tetanus, and 1 of "exhaustion," whatever this may mean, giving a lethal percentage of 3.7.[1] It is necessary to add that all of these were uncomplicated cases, operated on at a time when no septicæmia or pyæmia had set in yet ; the 57 cases of the latter order being set apart, as they ought to be, in a distinct table. Here we have 17 deaths versus 40 recoveries, a brilliant showing, considering the exceedingly grave septic processes that had necessitated operation. If all operations done by Prof. Volkmann in five years are thrown together, we see that he has lost 28 out of 318 cases, just as many as Prof. Ashhurst lost out of 100, which means that the Philadelphian's losses proportionately treble those of the German surgeon. Woelfler, of Vienna,[1] supplements this sufficiently conclusive proof in a still more convincing manner. He compares the mortality of Prof. Billroth's uncomplicated cases of amputation done in the same wards of the same hospital, with the same regulations for ventilation and cleanliness, in two numerically very similar groups of cases, operated on in consequence of similar indications, with an exactly identical technique of procedure, *but with a changed manner of dressing*, and demonstrates that whereas the first group of 58 cases (1871–1876) showed a mortality of 16 (21 %), the 56 cases of the second group (1877–1880), treated by the antiseptic method, all got cured, *not one of them died.* Of 704 amputations of all kinds done within the last decade or so by nine German surgeons, 110 were lost, representing a mortality of 15.6 %, a little less than a half of the author's average gained from the amputations of twelve English, French, and American sources. It is noticeable that Prof. Ashhurst wholly, we would almost say intentionally, neglects the enormous clinical material published by German authors. But the battle has been fought and well won long ago, and we must refer doubters to the original sources of information on the subject.

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[1] Oberst, *loc. cit.*

However meritorious Prof. Ashhurst's article may be, it is undeniable that his views and supporting statistics are far behind the rapidly striding times. Newer achievements are simply denied, and the only supports of this negation are a few sarcastic phrases, and a systematic ignoring of recent publications on the subject.

The special amputations of the extremities are very well set forth in a clear and precise manner, and will be found an excellent aid for reference. The hip-joint has received a very exhaustive treatment, the subject fitly terminating in a complete tabulated synopsis of known cases up to the date of publication. A large number of moderately well executed wood-cuts accompany the article. [A. G. G.]

Minute Structure of the Central Nervous System of Certain Reptiles and Batrachians of America. Illustrated by permanent photo-micrographs. By JOHN J. MASON, M.D. Series A. Author's edition one hundred. Newport, 1879–1882, pp. 31 ; 113 plates.

This elegant and valuable work is entitled to rank first among the neurological contributions which have been issued by the aid of photo-micrography. Indeed the volume seems as near perfection as the arts of printing and photography could make it; evidently no expense has been spared in its publication. It consists of about thirty pages of letter-press, and one hundred and thirteen plates, all photo-micrographs, which are beautifully executed, and, with a few exceptions, remarkably distinct in their details. Those alone who have had experience in the photographic representation of nervous tissues know the difficulties which prevent a satisfactory result in such work: difficulties of illumination and of focusing over a large field, not encountered when small anatomical elements are the objects to be photographed, to say nothing of the labor involved in obtaining sections suitable for the purpose, and a host of minor details. When all this is considered, Dr. Mason's contribution must be regarded as an extraordinary work, and one which could only have been completed by ardent devotion to his subject, involving extended labor, exceeding care, and great expense. The satisfactory results, as evidenced by these beautiful photographs, are, no doubt, a sufficient reward to the author; they cannot fail to be appreciated by all who have any interest in comparative neurology or photography. The production of a work of art is, however, a secondary object with the author; his aim being to furnish a contribution to comparative neurology by a more accurate method than the usual one by means of drawings, where numerous details are omitted and many inaccuracies are introduced from misconception by even the most conscientious and painstaking artist. The author himself admits that "a photograph cannot often show all that can be discovered by more direct microscopic observation," but adds that, "with a judicious working of the fine adjustment, high authority has stated, and perhaps correctly, that a good photograph with a low power—say from 3 to 1-2 inch—is a better means of illustrating the anatomical structure of nervous tissue than hand-drawing." "To give the student enlarged and exact representations of hardened specimens, showing comparative form and dimensions, with as much of the structure as possible, has been the chief aim in view." In the list of animals studied, we find no less than nineteen representatives.

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[1] Die Amputationen an der Prof. Billroth 'schen Klinic, 1877–1880. Wien, 1882. Braumüller.

Valuable as is this collection of permanent representations of the central nervous system, its value to the neurologist would be greatly enhanced if more details could have been given, either by descriptions, diagrams, or photographs, concerning the general anatomy of the different animals studied, particularly respecting the skelital and muscular relations, as they furnish one of the principal keys to the correct interpretation of the functions of the nerve centres. We trust that this will be found practicable for the promised second series. As the author has preferred to make his text principally explanatory, having refrained from presenting many generalizations, reference to the controversies raised heretofore regarding some of the points mentioned would, perhaps, be out of place. It is but just to say, however, that after reiterating his previously enunciated law, that "the nuclei of the so-called motor cells of the central nervous system have, in the same individual, average diameters which are proportional to the power developed in the related nuclei," he adds: "No sympathy with any theory which claims to distinguish between motor and sensory cells is here implied, nor does the writer insist upon any special anatomical importance for the nuclei, as compared with the cell-bodies, beyond their more distinct and regular outline, which makes them very conspicuous, and hence well adapted to accurate micrometric observation. That the nuclei are the true functional centres of the nerve cells is at best an unproved hypothesis; but comparative measurements of these bodies are facts which seem very essential to the future understanding of their function."

[W. R. B.]

The Spinal Nerves. By A. H. P. Leuf, M.D. Brooklyn: F. B. O'Connor, Jr., 1882, pp. 61, 6 charts, 1 diagram.

If there be "no royal road to knowledge," there are at least by-paths which lead more surely and directly to the desired goal than the beaten track pursued by the generality of workers; they are found, however, by those only who are systematic in their search. Much aimless journeying and doubling are done upon the highway of science, with a loss of energy which, properly applied, would have carried the traveller far beyond the point he finally attains. The proper arrangement and classification of a multitude of facts carry us a long way toward the solution of problems concerning them; nor is mnemonics to be despised as an aid to the retention of knowledge. Charts and diagrams can be made to serve such purposes; even smoothing the pathway for the student of that so-called dry subject anatomy. Who renders the attainment of knowledge less difficult is a common benefactor, and Dr. Leuf, in presenting to the students of medicine the publication above-named, merits the gratitude of all who avail themselves of its aid. The author's own language in the introduction sufficiently explains the plan of the work. "The spinal nerves are treated in three different ways: in the text, by charts, and by a diagram. While studying, the diagram may be pinned against the wall, the charts laid upon the lap, and the text held in the hand, while an illustrated anatomy or atlas should be lying close by for reference. It would, however, be much better to replace the atlas in the closet by the cadaver in the dissecting-room. The topographical arrangement is such that the names of the largest nerves have the largest type, while the name of the divisions and sub-divisions of these are printed in small and still smaller type; the names of the smallest being in italics, thus facilitating reference. The text, except when it is superfluous, is arranged exactly like the charts, the only difference being that each name is accompanied by more or less of a description. The arrangement of the diagram of *all* the spinal nerves *on one plate* is unique, as far as I know."

The amount of labor on such an arrangement is greater than would at first sight appear, and the author gives evidence of having devoted more thought to his subject than is required for a mere compilation. Although the proposal to change old-established anatomical terms is usually considered rather audacious, yet few will consider the substitution of the term "thoracic nerves" for that of "dorsal nerves" as otherwise than a rational one.

The cheap form in which the work is issued brings it within the reach of all students, and if properly placed on the market, will render it a more extensive educator than the elaborate and artistic works of Flower and others which seldom reach the dissecting-room on account of their cost. [W. R. B.]

ORIGINAL OBSERVATIONS.

TWO CASES OF PELVIC ABSCESS TREATED BY ABDOMINAL INCISION. RECOVERY.

By PAUL F MUNDÉ, M.D.,
GYNECOLOGIST TO MT. SINAI HOSPITAL, NEW YORK.

Of the large number of cases of pelvic cellulitis and peritonitis which have come under my professional care, but a comparatively small proportion have gone on to suppuration, probably not more than ten per cent., and of these few, some burst spontaneously into the vagina, rectum, or bladder, and others were aspirated through the vagina, the removal of the pus (perhaps several times repeated) being in every instance followed by complete resolution of the exudation, and recovery.

In none of these cases of suppuration did the pus point toward the abdominal surface, with one exception of spontaneous perforation seen many years ago in the lying-in hospital at Wurzburg. During the present year, however, two instances have occurred in my service at Mt. Sinai Hospital, in which the pointing of the abscess toward the cutaneous surface of the abdomen indicated its artificial evacuation at that point, and in which a free opening of the pus cavity resulted in perfect recovery. As these cases are not very common, and one, indeed, presents other complicating features of unusual interest, I will briefly report them.

CASE I.—*Large extraperitoneal plastic exudation* (*cellulitis*) *in left half of pelvic cavity ; suppuration ; free opening of abscess in left groin ; drainage into the vagina ; accidental laceration of bladder, with escape of urine into vagina and through abdominal wound ; spontaneous closure of fistula ; complete recovery.*

M. G., married, æt. thirty years, admitted May 18, 1882. Mother of six children, the last, twins, seven weeks previously, after which she was seized with a chill, high fever, and severe pain in the left hypogastrium which continued for several weeks, part of which time she was delirious. About five weeks after confinement she first noticed a large swelling in her left groin, which, on admission, showed distinct fluctuation on palpation and bimanual examination. The general condition of the patient was very poor; she was exceedingly anæmic, occasionally slightly delirious; temperature, 101°; pulse, 110, very feeble. Hot poultices to the abscess; stimulants and quinine were ordered, and sufficient opium to relieve the constant pain.

May 22d.—Patient somewhat stronger, pain less; temp., 100.6°. The swelling somewhat larger, extending toward the median line over the bladder.

May 25th.—Aspiration and removal of two ounces of pus, and subsequent small incision, with evacuation of at least a pint of pus. A long sinus was discovered leading toward the bladder and across Poupart's ligament. This was opened to the length of three inches, and the wound syringed out and packed with carbolized cotton.

June 8th.—The patient's temperature rose to 104°; pulse, 120, evidently in consequence of imperfect evacuation of pus. A long probe revealed a sinus extending transversely one inch across the median line immediately above the pubis, another some two inches upward into the iliac fossa, and a third, bifurcated, down into the pelvic cavity so that the point of the probe could plainly be felt through the anterior vaginal wall. As it was evident that thorough drainage was imperative, the patient was chloroformed, and the sinuses were freely laid open on the finger and director. The wound measured at least six by four inches and four inches in depth. A long director was then passed downward and the anterior vaginal wall gently pressed out until the corresponding spot appeared at the vulva, when a small opening was made directly on the point of the director, and the latter pushed through. A fine rubber drainage tube was then tied around the director, and with a rapid motion drawn into the sinus and out of the abdominal wound. As I drew the tube through, I noticed a slight obstruction at the lower orifice of the wound, which immediately yielded, but simultaneously the wound filled with a clear fluid, which was readily seen to be urine, and which also gushed from the vagina through the drainage tube. Evidently the bladder had been adherent to the wall of the abscess, its coats had become friable by long contact with pus, and, perhaps, by plastic infiltration, and the slight force exerted in drawing through the drainage tube ruptured it. No attempt was made to ascertain the size of the fistula ; the drainage tube was left in place, the wound packed with carbolized cotton, and hot poultices ordered, to reduce the extensive infiltration about the abscess. The bladder was directed to be washed out three times daily with a solution of boracic acid, one drachm, to the pint of tepid water, through an elastic catheter, which was left in the bladder. Stimulants, *ad libitum.*

June 9th.—Temp., 101° ; pulse normal ; wound looking well.

June 10th.—Temp. during last three days between 101° and 102°, but the patient feels and looks stronger and better. The fluid injected into the bladder flows freely out of the abdominal wound.

June 22d.—Patient's condition has not been so favorable during the last few days, apparently in consequence of fresh exudation in the iliac fossa. As hot poultices had been continued all this time, the effect of ice in restraining this spreading of the exudation was tried and with great success. Within twenty-four hours the temperature fell to 98.5°, the pain ceased, and the hardness and tension of the abdominal walls disappeared.

The drainage tube was now removed, as the wound was doing nicely, but the injections of two-per-cent. carbolic solution into the wound and of borax into the bladder were continued, and Peruvian balsam was poured into the wound as a gentle stimulant. The patient slowly gained strength and flesh, and when I saw her, on my return from the country, early in August, had grown almost stout and looked perfectly well. The permanent catheter was worn for about six weeks, until it was noticed that the bladder injection fluid no longer escaped by the abdominal wound, when it was found that the fistula had entirely closed, and the bladder had regained its normal function.

Sept. 3d.—Abdominal wound almost entirely closed. Patient stout and well and anxious to return home. Bladder in perfect order. Discharged.

CASE 2.—*Cellulitis in left broad ligament ; abscess ; free abdominal incision ; recovery.*

A. L., thirty-three ; married ; admitted, July 10, 1882. Tedious confinement four weeks previously. On tenth day had a chill followed by high fever and pain in left inguinal region and pelvic cavity. Two weeks before admission, noticed a slight swelling at seat of pain, which is perfectly distinct on admission, and on palpation and bimanual examination shows fluctuation. In the left broad ligament can be felt a large mass, evidently parametritic deposit. Temp., 101.4°.

July 11th.—Abscess opened by small abdominal incision, and about eight ounces of pus evacuated. A sinus was discovered leading toward the symphysis pubis, near which point a counter-opening was made, and a drainage tube introduced. Poultices and tonics were ordered. Temp. normal next day.

July 17th.—Patient better, but sinus gives no evidence of healthy action although washed out daily with two-per-cent. solution of carbolic acid. Appetite good ; temp. normal.

Aug. 4th.—As the sinus still refused to close, and was discharging thin, serous fluid, it was thought best to lay it thoroughly open, which was done by knife, on a director, inflicting a wound some six inches in length. There was considerable arterial hemorrhage, since the superficial epigastric and several small twigs were divided. Considerable difficulty was experienced in seizing the bleeding arteries because they were so deeply seated in the infiltrated tissue, but finally the larger branches were tied, the wound was washed with five-per-cent. solution of carbolic acid, and tightly packed with sheet lint, when all hemorrhage ceased.

Aug. 5th.—Patient doing very well. Poultice discontinued, and balsam of Peru poured into the wound, which was packed with dry oakum.

Aug. 11th.—Wound rapidly filling by granulation ; patient much stronger. Parametritic deposit is still hard, but smaller than when admitted. Tonics.

Aug. 24th.—Wound almost healed and deposit gradually disappearing.

Sept. 10th.—Wound entirely healed ; exudation only as large as a small walnut, but still hard. Patient feels very well. Discharged.

I do not propose entering into a lengthy discussion, either of the etiology or pathology, or of the principles of treatment, of pelvic abscess. I would merely say, that whenever such abscesses point toward the abdominal surface, they should be opened and treated precisely like similar collections of pus in other parts of the body. When there are several sinuses and openings, it is always best to split the former and unite the latter, even though the wound thus made may appear very large, since thorough drainage and cleanliness of the cavity, and rapid healing are undoubtedly promoted by such free incisions. When fluctuation can be felt with equal or even greater distinctness from the vagina, I think it unquestionably the better practice to aspirate, or, if necessary, incise there, since a cutaneous wound is avoided, and natural drainage is thereby at once established.

As regards the accidental production of a vesical fistula in case 1, I hardly see how it could have been foreseen. Still, in a future similar case, I should endeavor to avoid the accident, making the counter-opening at once so large as to easily admit the drainage tube. The spontaneous closure of the fistula was due to the constant withdrawal of the urine through the catheter, and to the frequent cleansing, and antiseptic injections made by the house-surgeons, Drs. Cramer and Rice, to whose unremitting care, in a great measure, the recovery of the patient is due. Relying on the statement of Dr. Emmet, that an artificial vesical fistula (such as is made as a cure for chronic cystitis) can scarcely be kept from spontaneous closure, I had from the very first assured the patient that the rent in the bladder would close without operation, although, I confess, that I did not feel quite as sure of that result as I pretended to be.

CASES ILLUSTRATING THE ACTION OF CONVALLARIA MAIALIS ON THE HEART.*

By ANDREW H. SMITH, M.D.

CASE 1.—A young lady, aged sixteen, in the third week of typhoid fever. An irritable stomach had made efficient alimentation difficult from the first, and although the temperature was never extreme, reaching only once to 104½°, and varying from 101½° to 103½° a day, adynamia occurred early, and the pulse showed a frequency out of proportion to the temperature, ranging between 120 and 140. The respiration was also frequent, not falling below 30 after the first week, and toward the beginning of the third week rising to 40. Repeated examinations of the chest failed to elicit any morbid physical signs.

Brandy freely administered produced remarkably little effect either upon the circulation or the sensorium.

Nov. 15th, being toward the close of the third week, and two days before death, the pulse was 144 in the morning, and by the time of my visit at 6 P.M., had reached 150, and was so feeble that it was very difficult to count it accurately. The first sound of the heart, though distinguishable, was very faint. Five minims of the fluid extract of convallaria, prepared by Parke, Davis, & Co., had been given three or four hours before, but it had been immediately vomited. At six o'clock, four minims were administered hypodermically. At nine o'clock the pulse was still 150, but notably fuller and more distinct. The trained nurse stated that she observed a change in the quality of the pulse about an hour after the injection was given. The respirations were 50 per minute and very superficial. First sound of heart very faint. Lips blackened with sordes ; face dusky.

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* Read before the Section on Practice of the Academy of Medicine, Nov. 21, 1882.

Dr. C. W. Packard, who was present in consultation, having concurred in the propriety of repeating the dose, six minims were injected at 10 P.M. At 10:15 the pulse had fallen from 150 to 128, and was distinct, but irregular. At 10:20 it was 120, strong and regular. Five minutes later it was 108, and soon after it touched for a moment 98. Both heart-sounds were almost preternaturally loud, and the cardiac impulse was plainly felt. The respiration, however, remained at 50, but was somewhat deeper. The face lost its dusky hue and assumed a fresh and natural color. There was no appreciable effect upon the sensorium, the delirium continuing as before ; subsultus and picking at the bedclothes, which were present, also remained unchanged. At 10:35 the maximum effect was obtained. The pulse from that time began to rise again, and at 11 o'clock was 126. The respirations increased to 60. From these figures there was again a decline, and at 7 o'clock the next morning the pulse was 102 and the respiration 48. The sordes had disappeared from the lips. The temperature showed but little change, remaining between 102 and 103. There was a good deal of nausea during the night, and several attempts to vomit. The urine, which all along had been abundant, was not increased.

The local effect of the injection was limited to the production of a small erythematous patch, which remained until death.

In the course of the day the pulse increased to 120, but still retained a fair amount of force. During the night, however, the coma deepened, and it became more and more difficult to administer nourishment or stimulants. Mucous râles developed in the lungs, the radial pulse could scarcely be distinguished, and by 7 o'clock in the morning of the 17th the patient was plainly moribund. Yet, even then, both heart-sounds were clear and distinct. Death took place at 9 o'clock A.M.

It would seem that the action of the convallaria, in this case, at least, was the reverse of what is observed when the pneumogastrics are divided. This operation results in quickening the heart's action and slowing the respiration, while the convallaria slows the pulse and quickens the breathing.

This case confirms, in the main, the observations of Prof. See and those made in 1867 by Dr. Marmé, of Göttingen.[1]

CASE 2.—Called, in consultation with Drs. A. S. and A. E. M. Purdy, to see a patient at the Windsor Hotel, Nov. 20, 1882, at 7:30 P.M.

The case was one of peritonitis, in a gentleman about 35 years of age. He had been taking a grain of opium every two or three hours, and at half-past four was bright and intelligent when spoken to, showing nothing more than a slight drowsiness. At six he was found in a stupor, from which he could not be roused. Dr. Purdy was called, and found him unconscious, with limbs rigid, a good pulse, and breathing fourteen times a minute. Respiration, however, soon became very superficial, and in the course of a few minutes apparently ceased. Artificial respiration was resorted to, and a battery was employed. Breathing was restored after nearly half an hour's labor, but it was very shallow, catchy, and irregular. At this time I saw the patient. He was breathing so imperfectly that compression of the chest was frequently required to excite a respiratory effort, which would then be followed by half a dozen quick, jerky superficial respirations. The face was livid, the mouth open, conjunctivæ scarcely sensitive, pupils moderately contracted; pulse very rapid and irregular, at times large and gaseous, then flickering and scarcely perceptible. The heart's action was a confused wobbling, in which no distinction of first and second sound was possible.

Although the picture was so little that of opium poisoning, it was thought well to administer atropine and coffee, as both are heart tonics, and were indicated irrespective of the question of opium narcosis. Atropine and caffeine were given hypodermically, and half a pint of black coffee was thrown into the rectum. No result having been obtained in the course of three quarters of an hour, the symptoms showing no improvement, it was determined to try the effect of convallaria. Eight drops were injected under the skin at 8:30, the pulse then being 150 and exceedingly feeble; the respiration varying from 30 to 44; the inspirations quick and sibilant, the expirations moaning. Face deeply livid; lips and tongue almost black. A specimen of urine was obtained by passing a catheter, and was found to contain albumen. At nine o'clock the condition was unchanged, and eight drops more were injected. In the course of fifteen minutes the pulse fell to 135; five minutes later it was 125; at the next counting it was 120, full, strong, and regular. The respirations became fuller, and slower, and more regular ; the lividity gradually disappeared from the face ; the lips became red ; the eyelids moved when touched ; the mouth closed when a spoon was put to the lips ; soon the hands were moved ; the eyes opened when the head was lifted up ; intelligence began to return, and when asked by his mother if he knew her, he replied yes. By half-past eleven consciousness was fully restored, and the patient was able to converse.

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[1] *N. Y. Med. Journal*, Nov., 1867.

As the action of the heart grew more steady, a harsh murmur was heard with the first sound at the base. On inquiry of the family, it was ascertained that for some time he had been short of breath on going up stairs.

At nine o'clock this morning (21st), barring headache and some abdominal tenderness, he was perfectly comfortable. Pulse 108, temperature 101$\frac{3}{5}$°.

TWO CASES OF GLYCOSURIA ; ONE TRUE AND ONE SIMULATED.

By E. C. SEGUIN, M.D.

The two following cases are presented because they are both in some way unique.

Case 1.—Mrs. —— consulted me in October of this year for simple, moderate dementia. She was fifty-one years old, and had masturbated for years. Six years ago she had an attack of quite acute melancholia.

In the last three years marked emaciation had taken place. No positive delusions or hallucinations seemed to exist. The memory had been fairly retained.

The patient fancied that her shoulders were paralyzed, and wanted to have her arms cut off. There existed no actual paralysis. The patient's manner was demented and hypochondriacal. She had no symptoms of diabetes except the emaciation, hence it was not suspected.

As a matter of routine her urine was examined with the following result. An afternoon specimen had the specific gravity of 1045, and contained a trifle over five per cent. of sugar, but was otherwise normal. A specimen passed the next morning had the specific gravity of 1018, and did not contain a trace of sugar.

October 14th.—Two days later, a morning and night specimen were sp. gr. 1020 and 1011 respectively, and contained no sugar.

Oct. 16th.—The urine had the sp. gr. of 1019 and 1023, and was free from sugar.

Oct. 21st.—Four specimens were found of normal specific gravity and reaction. When last heard from (about November 12th) the patient was doing well and had no symptom of diabetes. The patient had neither medication nor diet which could have caused the sudden disappearance of sugar from the urine.

Cases of mild diabetes are not rare where an occasional sample of urine is found devoid of sugar ; but a fall in the specific gravity from 1045 to 1018 and from five per cent. of sugar to none in twenty-four hours, is by no means common.

How long the patient had been passing sugar cannot be told, and we may perhaps look on the case as one where some peripheral sensory impression or some transient central alteration produced a disturbance of the chylo-poietic circulation which interfered in proper glycogenesis. This is rendered more probable because Pavy, Cyon, Aladoff, Schiff, and Eckhard have shown that itritation or destruction of many parts of the peripheral nervous system will, in a reflex way, produce glycosuria, while by puncturing the floor of the fourth ventricle, Bernard long ago caused sugar to appear in the urine of animals. Among diabetics, too, a profound mental or bodily shock almost invariably causes an increased excretion of sugar.

Case 2.—In May of this year an old patient of mine sent three specimens of urine, with the request that they should be thoroughly examined. He said they were passed by a niece of his, who was under the care of a homœopathic physician for some hysterical affection. More definite data than these it was difficult to obtain. A night specimen of May 18th was pale, cloudy, contained some uric acid, and a *trace* of sugar. It had the sp. gr., however, of 1070 ! A specimen passed the next morning contained only a trace of sugar, and had the sp. gr. of 1048. A noon specimen of that day was pale, poured like syrup, and, with only about three per cent. of sugar, had the sp. gr. of 1090 !

Besides these examinations, which were made by Dr. Amidon, I sent the heavy specimen to Mr. Charles Rice, of Bellevue Hospital, and the following is an extract from his letter in reply :

" There is, however, something present, which I cannot make out exactly, but which I have separated and find to resemble in some of its properties ' peptone,' or digested albumen. If a portion of the urine is mixed with about five times its volume of absolute alcohol and shaken, a gummy substance separates, which may be caused to agglutinate into a lump by judicious turning of the test-tube. After being washed with alcohol it is found to be but little soluble in boiling water, but it dissolves in cold water, particularly after addition of a drop or two of hydrochloric acid, to a very thick, almost colorless, faintly opalescent liquid. It gives no reactions with nitrate of silver, or mercuric chloride, and does not exhibit any reactions of the ordinary gums."

June 6th.—Two more specimens were sent and examined by Dr. Amidon and Mr. Phelps. A night specimen was clear, pale, and gave a faint sugar reaction. It was syrupy, and had a sp. gr. of 1105! A morning specimen, while it seemed to contain less sugar, had a sp. gr. of 1117!! A fraud was immediately suspected, and its detection was attempted.

Mr. Rice kindly examined the latter specimen and said: "The last specimen of 'urine' seems to contain but little genuine urine, to judge from the small amount of the sulphates, phosphates, chlorides, and urea present." "It contains a body (or mixture of bodies) insoluble in alcohol, and partaking of *some* of the properties of starches and of gelatin, without, however, giving a reaction for starch itself, or a definite reaction for gelatin." "I should say the thing is a fraud."

A series of experiments were then made with gelatin, cane sugar, and various syrups. It was finally found that a mixture of a fine sorghum syrup and urine, in such proportions as to bring the specific gravity up to about 1115, produced an exact imitation of our puzzling specimen. It gave the same precipitate to absolute alcohol, and contained the same amount of grape-sugar. It was, moreover, found by Dr. Amidon, that on standing and fermenting the cane sugar was transformed into glucose and gave a magnificent reaction to Fehling's test, while, when fresh, only a faint sugar reaction was obtainable. Also it was noticed that the specific gravity of the specimen of June 6th, which had stood in the laboratory uncorked for ten days, had fallen from 1117 to 1010, showing that the cane sugar had fermented and disappeared.

The mother of the patient was asked to *see* some urine passed in a clean vessel and send that for examination.

It was some time before this could be brought about, but at last a night specimen of urine was sent in which was clear, of a reddish-yellow color, acid, free from albumen and sugar, and of the specific gravity of 1024.

NOTE.—Copies, on heavy paper, of the cuts in Dr. Seguin's article, page 260, can be obtained of the publishers.

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